Cardiology

Version 5.3

Corrected, Updated, Lighter

PLAB 1 Keys is for PLAB-1 and UKMLA-AKT (Based on the New MLA Content-Map)

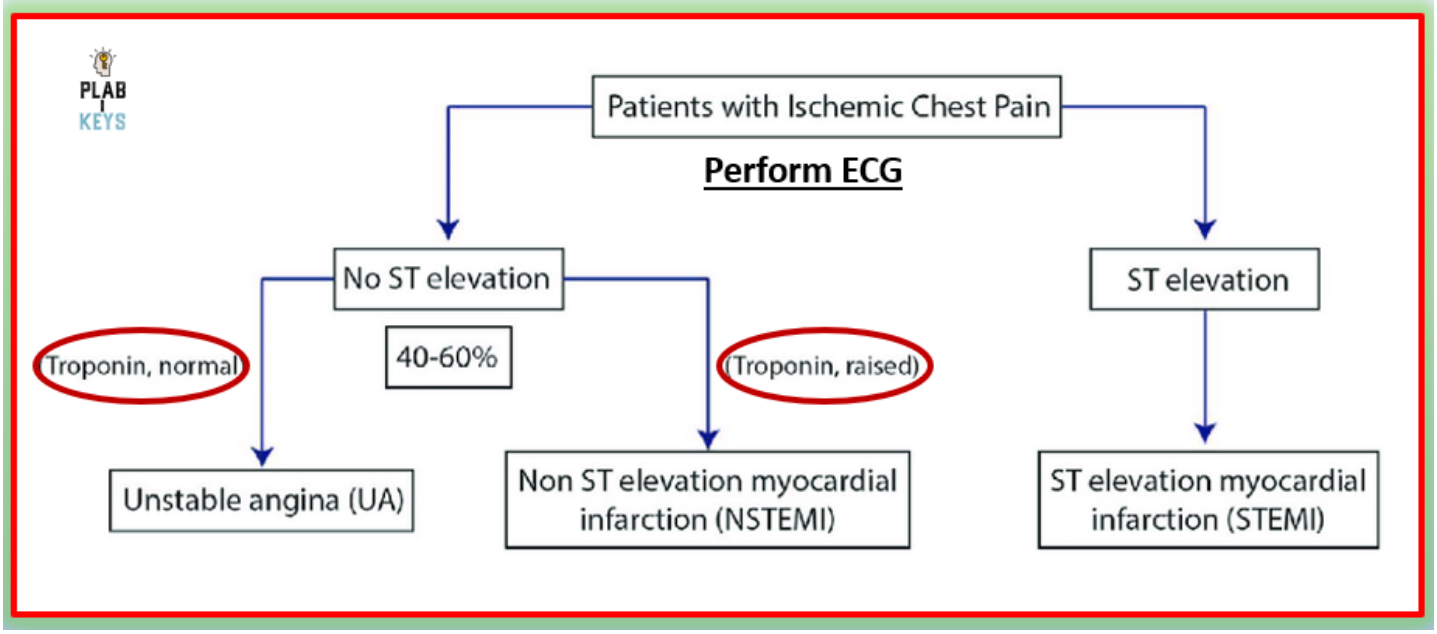
With the Most Recent Recalls and the UK Guidelines

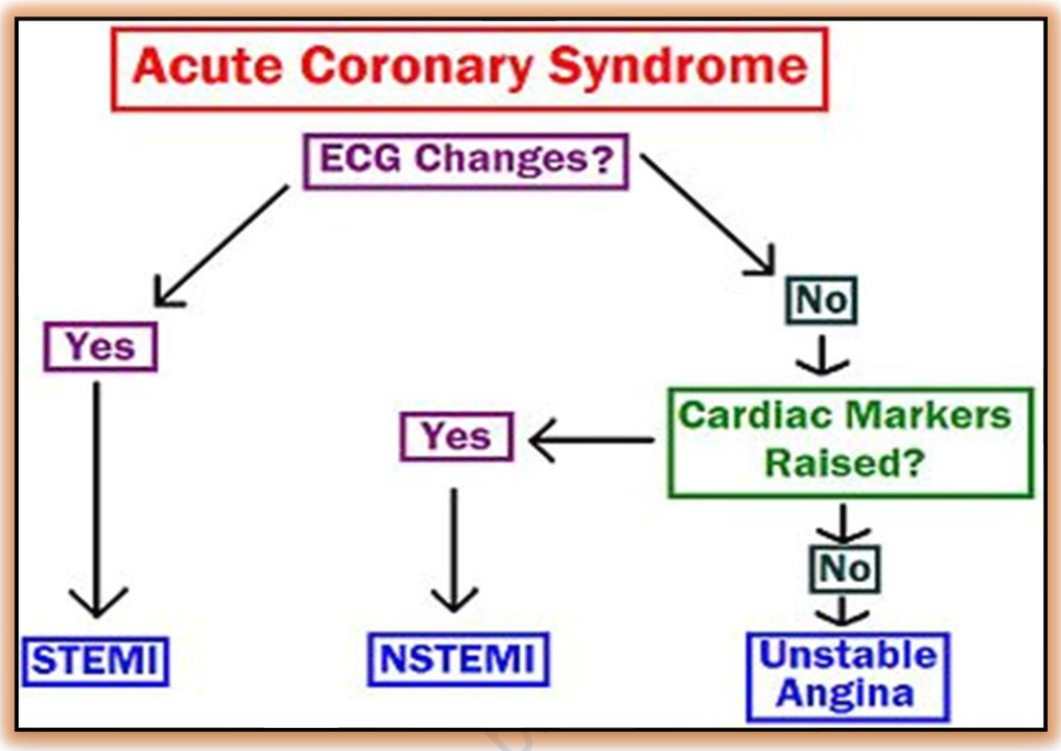
ATTENTION: This file will be updated online on our website frequently!

(example: Version 2.2 is more recent than Version 2.1, and so on)

Key 1

Acute Coronary Syndrome (ACS)





◙ Acute Coronary Syndrome includes:

√ ST elevation myocardial infarction (STEMI).

√ Non-ST elevation myocardial infarction (NSTEMI).

√ Unstable angina.

◙ Symptoms and signs

The classic and most common feature of ACS is **chest pain**.

√ Typically, **central**/**left-sided**/ **substernal**/ **epigastric**.

√ May radiate to the **jaw**, the **left arm**, the **shoulder**.

√ Often described as **'heavy'** or **constricting**, 'like an elephant on my chest'

- It should be noted however in real clinical practice that patients present with a wide variety of types of chest pain and patients/doctors may confuse ischaemic pain for other causes such as dyspepsia.

- Certain patients e.g. **diabetics**/elderly may **not** experience any chest pain

→ **Silent MI**

◙ **Other possible symptoms in ACS include:**

Dyspnoea ▌sweating ▌nausea and vomiting ▌may appear pale and clammy

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◙ Risk Factors of Ischemic Heart Disease:

Unmodifiable risk factors	Modifiable risk factors
Increasing age Male gender Family history	Smoking Diabetes mellitus Hypertension Hypercholesterolaemia Obesity

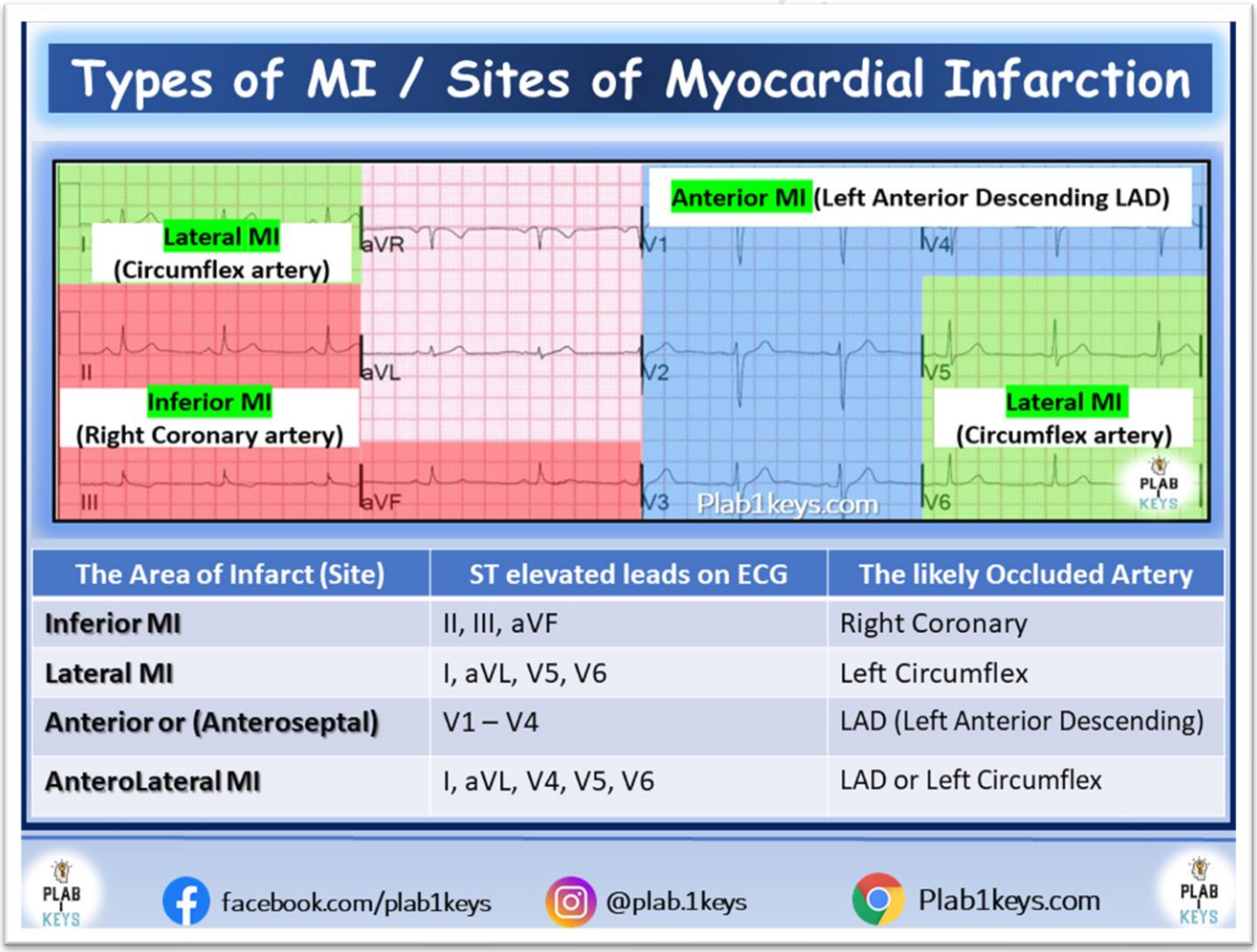
◙ Investigations

√ **ECG**

√ **cardiac markers** e.g. **troponin**

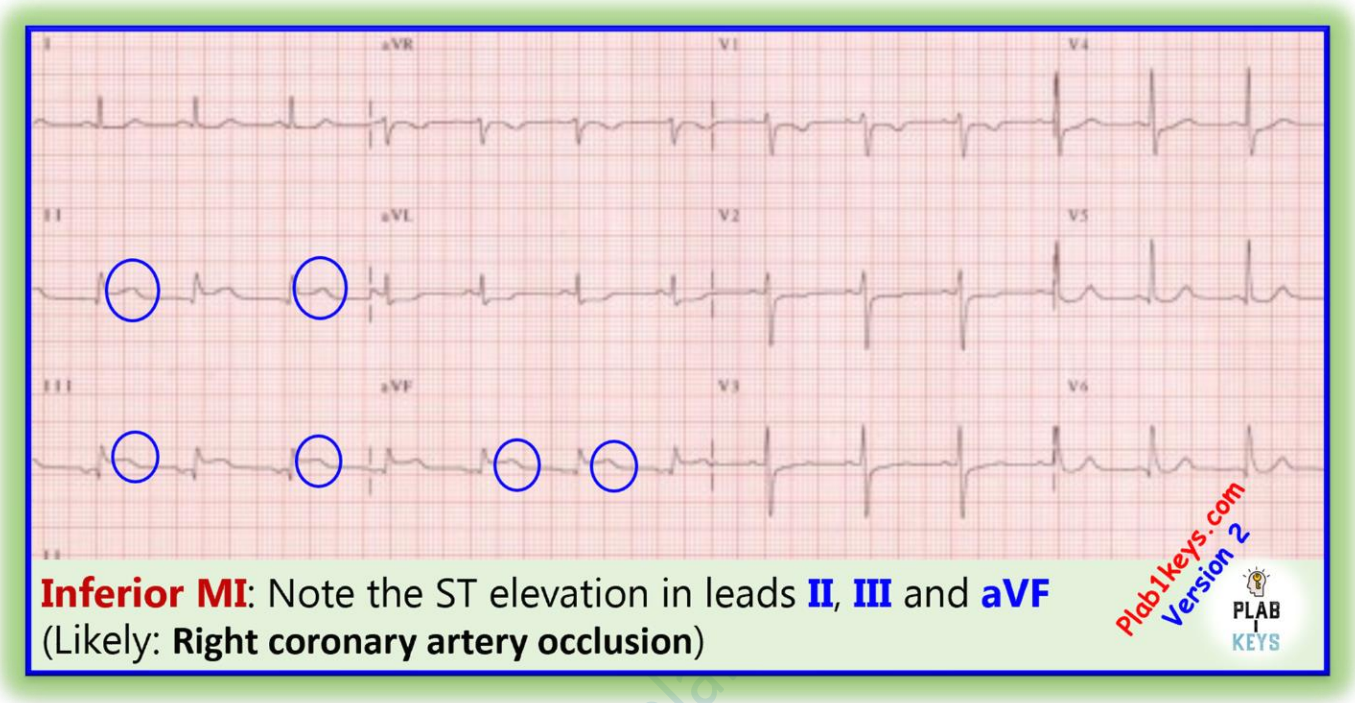
ECG in ST-elevation MI

→ elevated ST segment in certain leads gives a clue about the site and type of the STEMI as follows:



The Area of Infarct (Site)	ST elevated leads on ECG	The likely Occluded Artery
Inferior MI	II, III, aVF	Right Coronary
Lateral MI	I, aVL, V5, V6	Left Circumflex
Anterior or (Anteroseptal)	V1 – V4	LAD (Left Anterior Descending)
AnteroLateral MI	I, aVL, V4, V5, V6	LAD or Left Circumflex

Example:



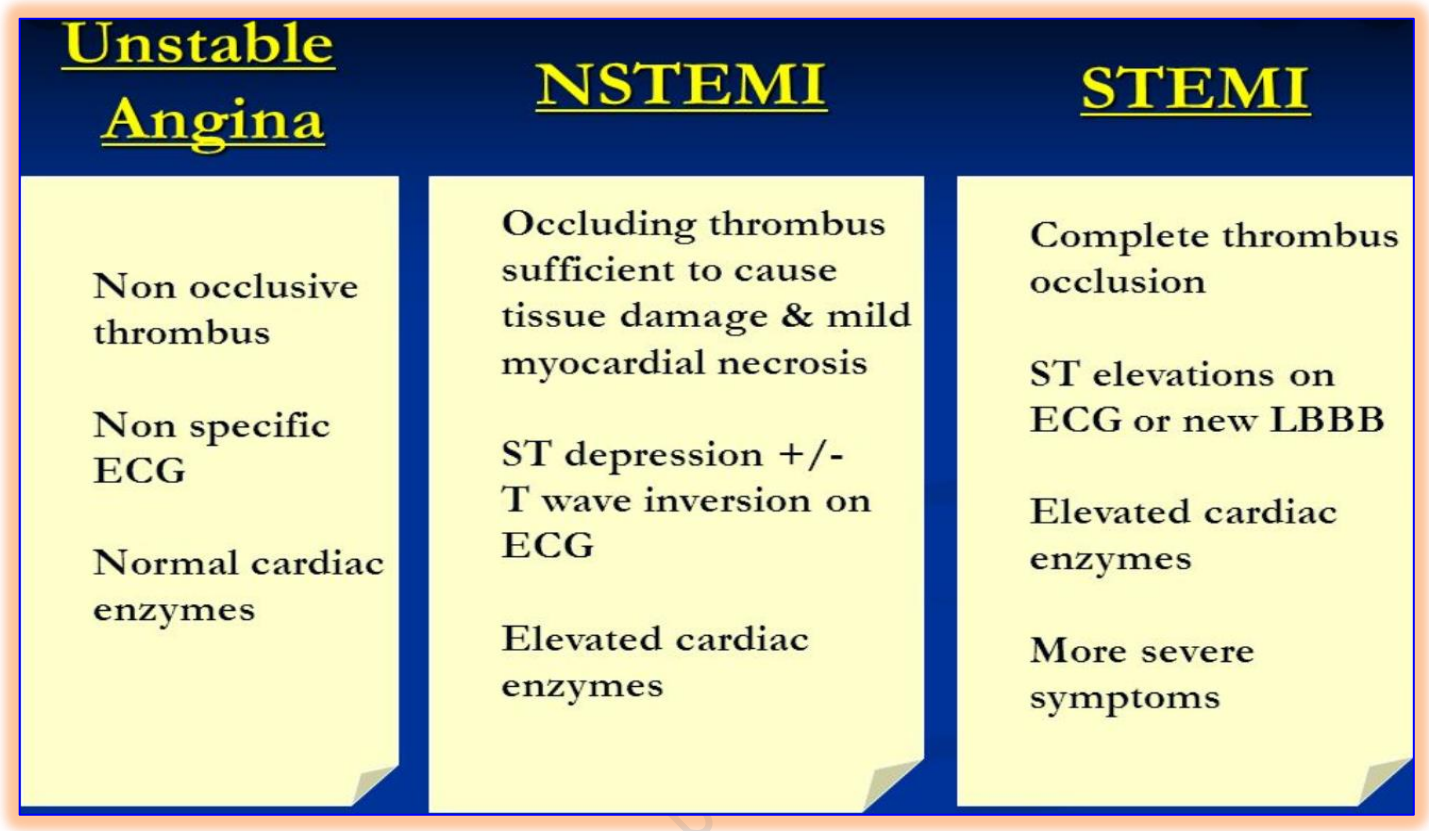
Inferior MI: Note the ST elevation in leads **II**, **III** and **aVF**
(Likely: **Right coronary artery occlusion**)

ECG features of **Left main coronary artery occlusion** [LMCA]:

√ **Wide spread ST depression**.

√ **ST elevation in aVR**.

Do → **Emergency coronary angiography**.



Management of ST elevation MI (STEMI):

◙ **In Acute Settings** → **MONA**

(IV Morphine, O2, Nitrates, Aspirin 300 mg)

- If the patient presents **within 12 hours** of the onset of the symptoms

  → Primary **PCI** (**P**ercutaneous **C**oronary **I**ntervention) **"The gold standard"**

In this procedure (**PCI**), the blocked arteries are opened up using a balloon (angioplasty) following which a stent may be deployed to prevent the artery occluding again in the future. This is done via a catheter inserted into either the radial or femoral artery

- **If Not, or PCI is unavailable**

  → Thrombolysis **(Alteplase is preferred over Streptokinase).**

- **(Chronic) Long-term Management of MI:**

1) Aspirin **for life.**

2) Ticagrelor **or** Prasugrel **for 12 months "**or: Clopidogrel**".**

3) Beta Blockers **(for 12 months)** "e.g. atenolol, bisoprolol ® *concor, zebeta".*

4) ACE inhibitors (**for life**) "e.g. captopril, enalapril, ramipril"

[If intolerant to ACEi such as dry cough, use ARBs instead e.g. losartan, valsartan, irbesartan]

5) Statins (**for life**) "e.g. Atorvastatin 80 mg PO OD".

So, Long-term MI Rx = 5 Drugs: Aspirin, Clopidogrel, BB, ACEi, Statins

AABC+S → **A**spirin, **A**CE inhibitors, **B**eta-blockers, **C**lopidogrel + **S**tatins

Management of NSTEMI & Unstable Angina:

(based on the recent UK guidelines)

Important:

For all patients where the diagnosis of NSTEMI or Unstable Angina is made → Aspirin 300 mg (+) LMWH e.g. Enoxaparin, Dalteparin "or Fondaparinux" need to be given as soon as possible.

√ Aspirin 300 mg.

√ Nitrates or morphine to relieve chest pain if required.

√ Antithrombin: **LMWH e.g. Enoxaparin, Dalteparin "or Fondaparinux"** should be offered to patients who are not at a high risk of bleeding and who are not having angiography within the next 24 hours.

If angiography is likely within 24 hours or a patient's creatinine is > 265 µmol/l, unfractionated heparin should be given.

[**Note**: **Fondaparinux** and **LMWH** are given **Subcutaneously**, whereas **Unfractionated Heparin** is given **Intravenously**].

√ Second antiplatelet: e.g. Clopidogrel, Prasugrel.

√ **Intravenous glycoprotein IIb/IIIa receptor antagonists (eptifibatide or tirofiban)** should be given to patients who have an intermediate or higher risk of adverse cardiovascular events (predicted 6-month mortality above 3.0%), and who are scheduled to undergo angiography within 96 hours of hospital admission.

√ **Coronary angiography** should be considered within 96 hours of first admission to hospital to patients who have a predicted 6-month mortality above 3.0%. It should also be performed as soon as possible in patients who are clinically unstable.

Examples of recent exams' questions:

[Example 1]

A patient presents with (acute chest pain radiating to jaw and shoulder + other features suggesting ischemic heart disease…) However, without ST elevation on ECG. What to Do Next?

→ Measure Cardiac Enzymes, especially (**Troponin**)

√ If Troponin is high → **NON-STEMI = Non-ST elevation MI**

√ Immediate management

→ **Give Subcutaneous LMWH OR Fondaparinux** + **Aspirin 300 mg**

Notes:

LMWH = Low Molecular Weight Heparin

Examples → Dalteparin, Enoxaparin

Fondaparinux (trade name Arixtra) is an anticoagulant medication chemically related to low molecular weight heparin.

[Example 2]

A 60 YO man with Hx of smoking, HTN and DM presents to his GP complaining of 25 minutes of left side dull aching chest pain radiating to his jaw. He was given Aspirin 300 mg by his GP and then sent to medical services in a local hospital. He is no longer in pain. The ECG is normal. The troponin is elevated 202 ng/L (Normal: < 5 ng/L). What is the next step in management?

A) Alteplase.
B) **Subcutaneous fondaparinux**.
C) IV Glyceryl trinitrate (GTN).
D) IV Morphine.

Since the ECG is normal, alteplase is wrong.

Since **ECG is normal** and **Troponin is high** → **Non-STEMI**

→ **Anti-coagulation** (**LMWH** e.g. **Dalteparin**, **Enoxaparin** or **Fondaparinux**).

[Example 3]

A 62 YO man with Hx of smoking and HTN presents complaining of 25 minutes of left side constricting chest pain radiating to his left shoulder. He was given Aspirin 300 mg and trinitrates for the pain. ECG was then done and showed ST elevation in leads V1-V4. What is the most appropriate next step in management?

→ **PCI "Percutaneous Coronary Intervention"**.

If not among the choices, pick → **Alteplase "Thrombolysis"**

[Example 4]

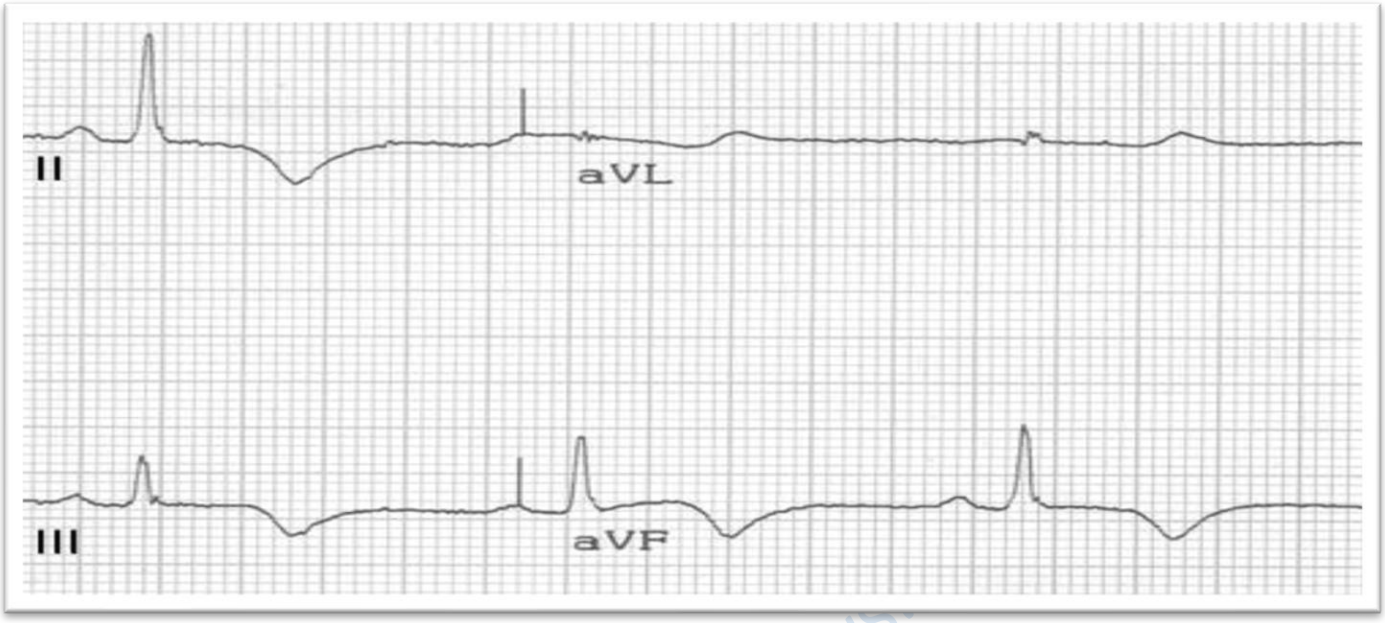
A 59 YO hypertensive patient presents to the A&E complaining of dull central chest pain for around 4 hours. His vitals are as follows:

HR: 99, BP: 155/95, RR: 21, O2 sat on room air: 97%

Chest X-ray is normal. Troponin level is pending.

He was given IV morphine for his chest pain.

The ECG is as follows:



What is the most appropriate next step in management?

Chest pain + T wave inversion suggests → myocardial ischemia.

In this case, 2 drugs should be given immediately:

√ **Aspirin 300 mg**.

√ **LMWH or Fondaparinux**.

Pick the one that is given in the choices.

"low-risk patients can be treated conservatively. However, if subsequent ischemia develops → **coronary angiography** with PCI".

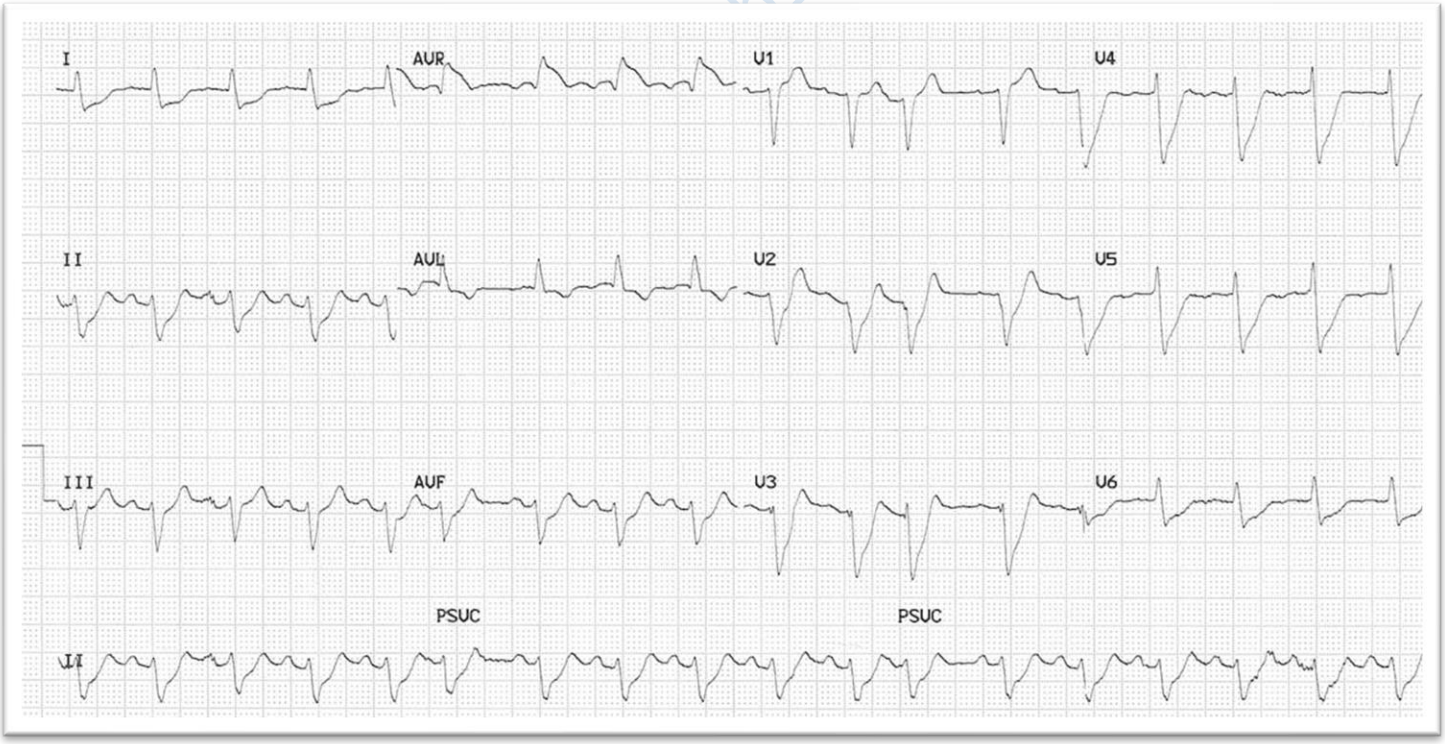
What if the ECG shows features of left main coronary artery occlusion (Wide spread ST depression + ST elevation in aVR)?

→ **Emergency coronary angiography**.

[Example 5]

A 61 YO patient presents to the A&E complaining of dull central chest pain for around 4 hours. His vitals are as follows:

HR: 75, BP: 135/85, RR: 21, O2 sat. on room air: 97% He was given IV morphine for his chest pain. The ECG is as follows:



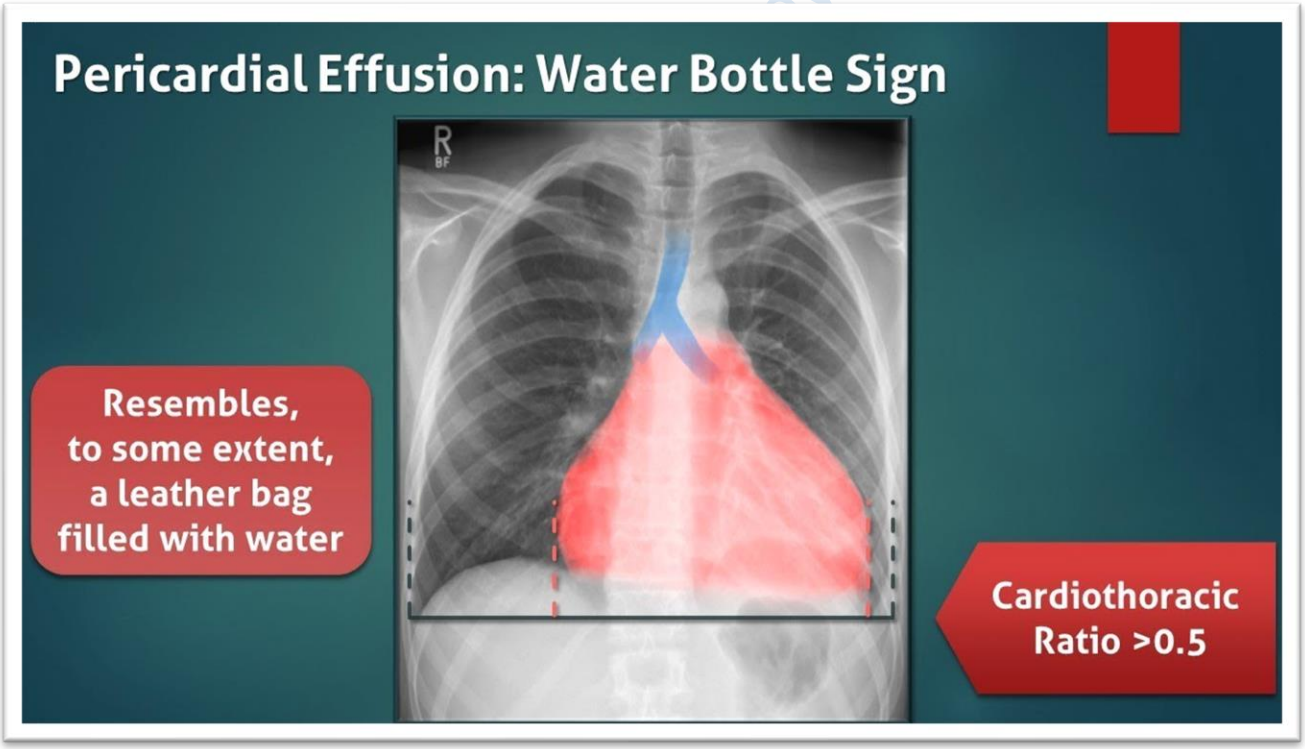
What is the most appropriate next step in management?

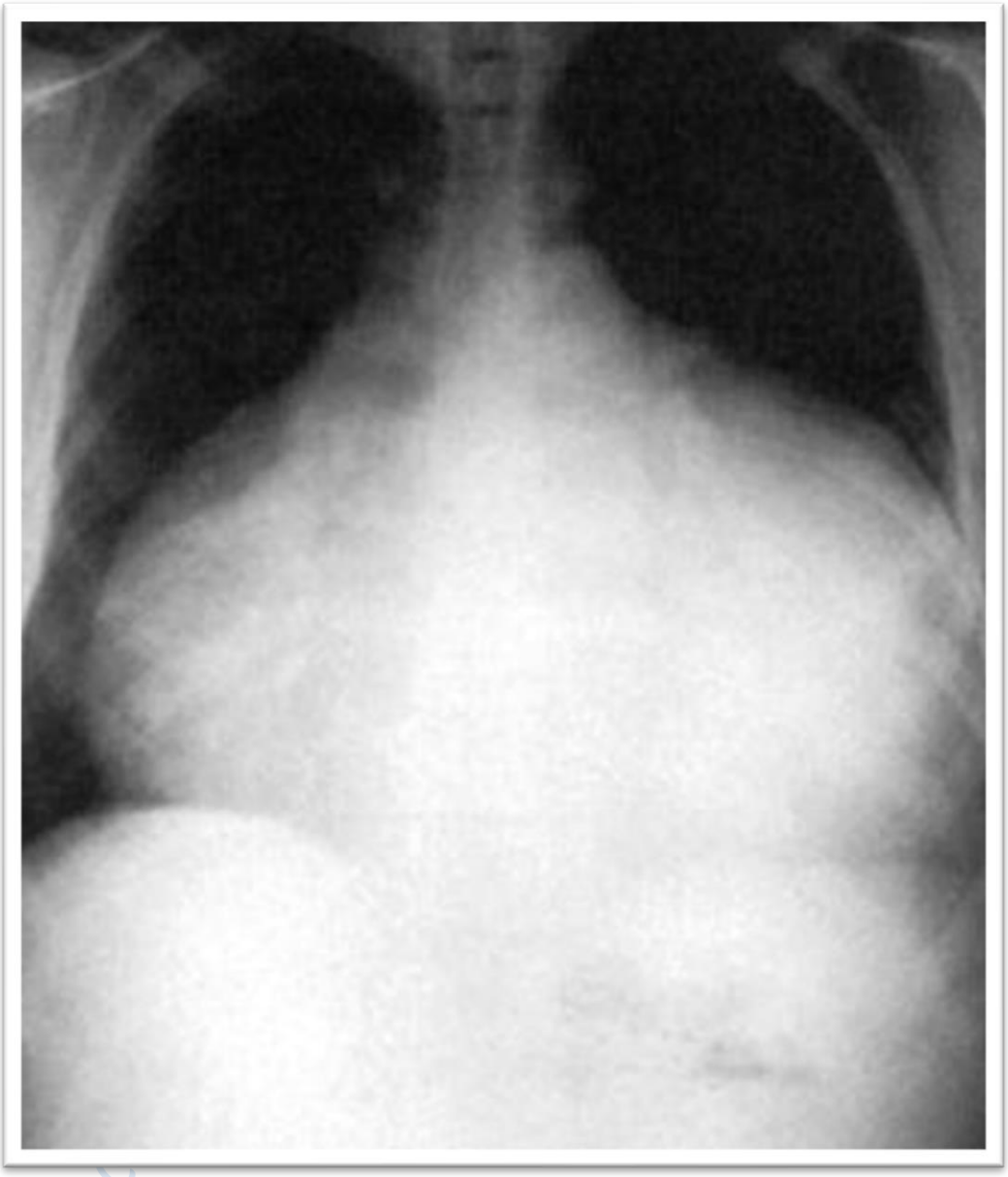
	This ECG shows the typical features of Left main coronary artery occlusion: • **Widespread ST depression**, and • **ST elevation in aVR**. Do → **Emergency coronary angiography**.
Key 2	**Cardiac Tamponade** **Accumulation of pericardial fluid under pressure** • **Beck's Triad**: **Hypotension ▌Muffled Heart Sounds ▌High JVP** (Distended neck veins). • **Others**: Dyspnea, Pulsus Paradoxus, Tachycardia. • **Cardiac Tamponade can develop as a complication of MI** After MI → Acute pericarditis → Pericardial effusion → Cardiac Tamponade • **Trauma** is the most important cause of cardiac tamponade.

N.B. **Chest X-ray** that shows **enlarged globular heart**

→ Think of either **Pericardial effusion** (**OR**) **Cardiac Tamponade**.

- **Dx**: **Echocardiogram** is diagnostic
- **Rx**: **Urgent pericardiocentesis**.

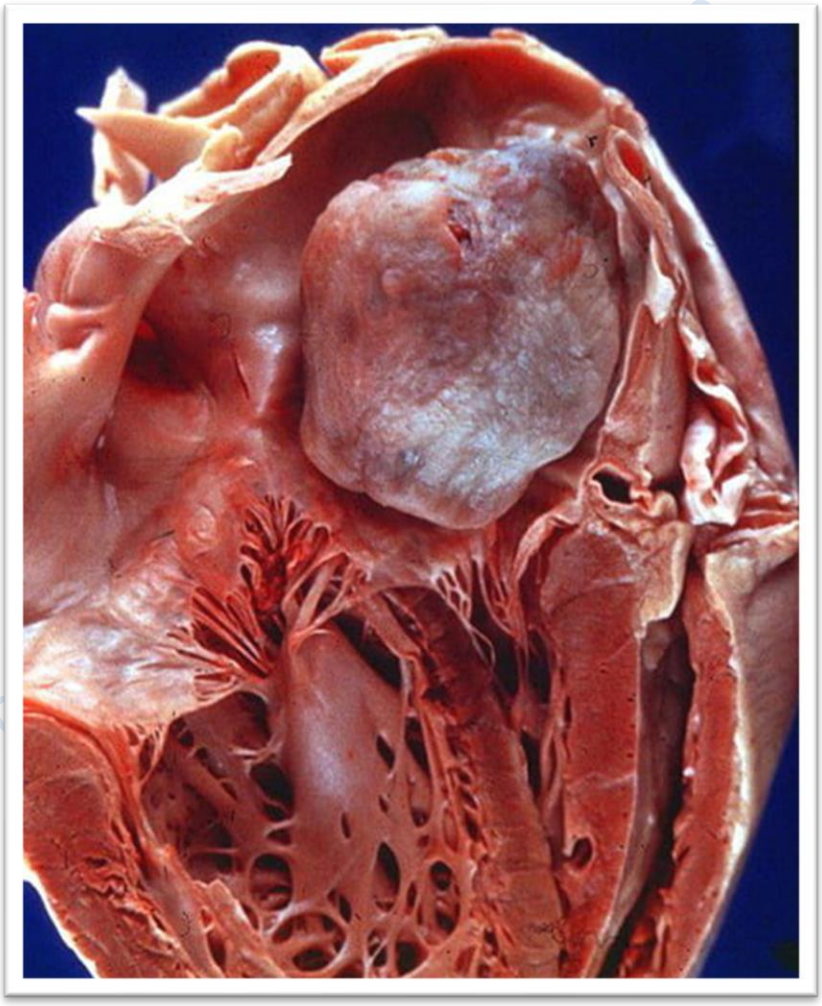




Cardiac Tamponade

Important!

If the patient is in hypovolemic shock (severely low BP) and the question asks about the [**INITIAL**] treatment line and **IV fluids** is within the option, pick it!

	Cardiac Tamponade: Oxygenation and ventilation → 1 to 2 L IV fluid NS → bedside Pericardiocentesis.
Key 3	**Atrial Myxoma**  ○ Benign tumours.

- 75% in the **left** atrium.
- Tend to grow on the wall (inter-atrial septum).
- 10% are inherited → Familial myxoma

Features:

◙ **Obstruction of Mitral valve** → **Mid-diastolic murmur**, **Dyspnea**, **Syncope**, Congestive HF.

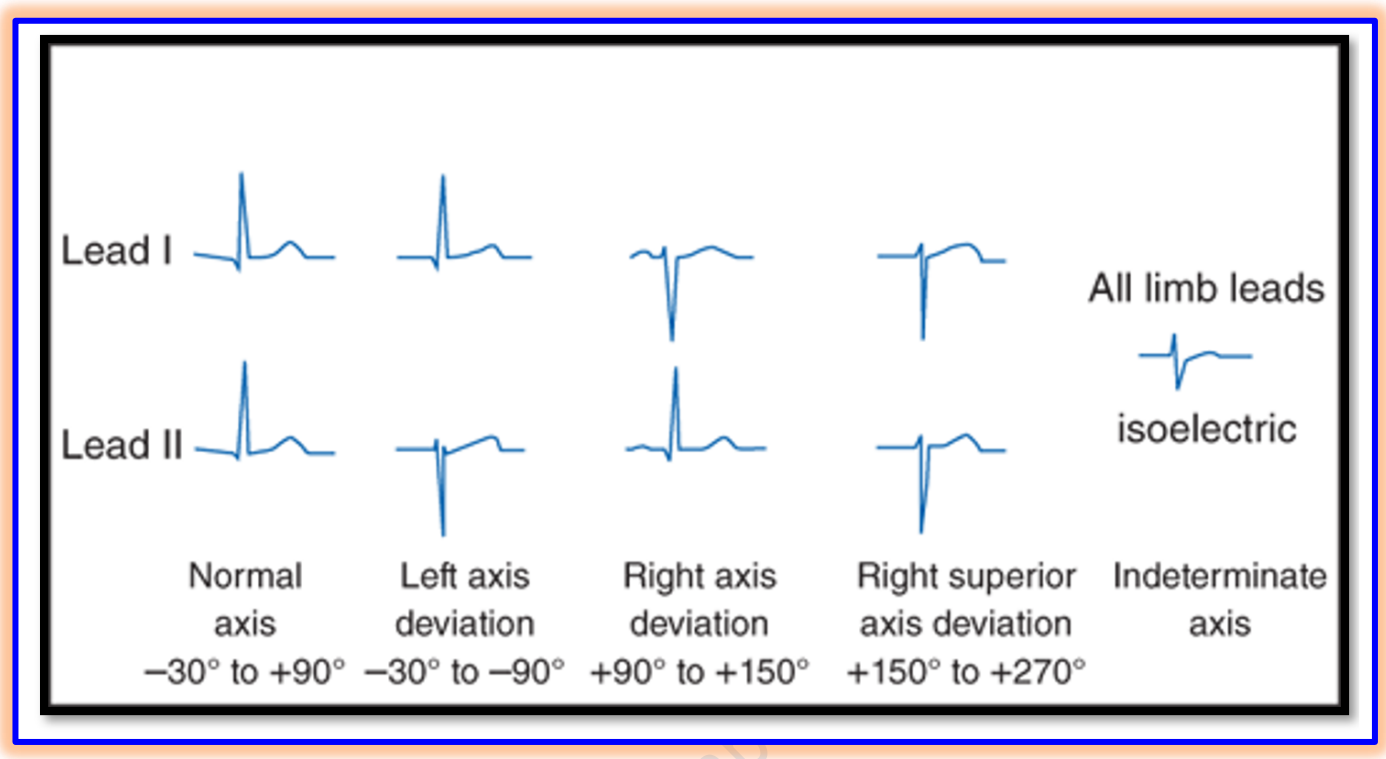
◙ **Small pieces may break off and travel to arteries** causing (**ischemia**) of different parts of the body such as:

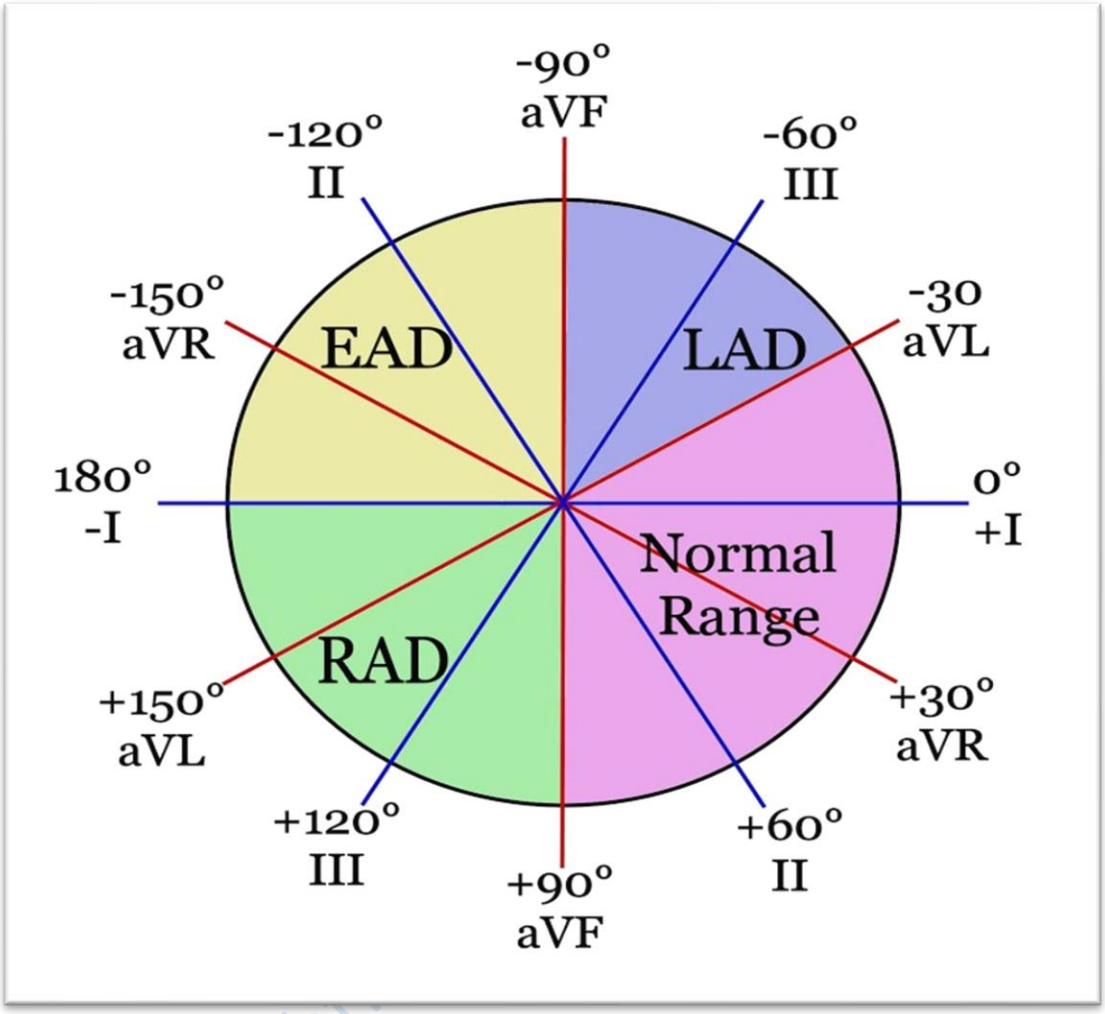
- ◆ **Lung** → Can cause **PE** (Pulmonary Embolism)
- ◆ **Brain** →Can cause **Stroke**
- ◆ **Peripheries** → **Clubbing** and **Blue fingers**.

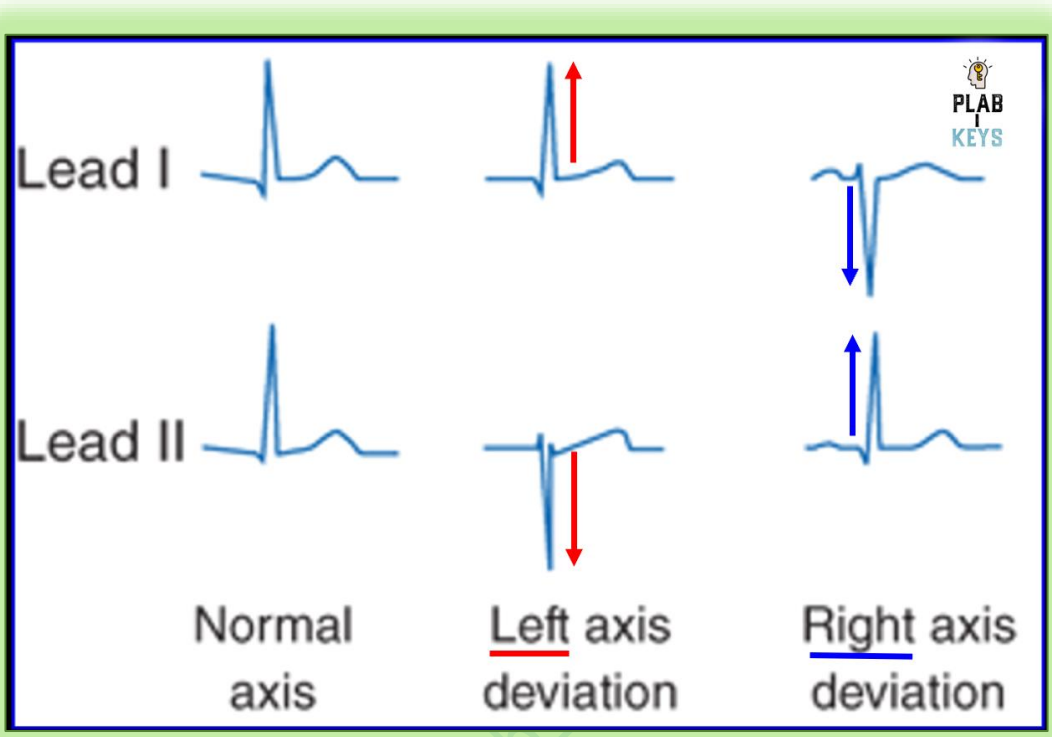
◙ **Atrial Fibrillation**

- Dx → **Echo** → Pedunculated heterogenous mass typically attached to the region of fossa ovalis (inter-atrial septum).
- Important note:

	If acute limb ischemia develops "sudden painful swollen limb with a loss of pulse" → we could save the limb by performing an urgent catheter embolectomy.
Key 4	**A patient was hit by a car into his chest and is brought to the emergency department. His neck veins are distended, Heart sounds are faint, hypotensive and tachycardic.** The likely Diagnosis → **Cardiac Tamponade**. The most appropriate management → **Pericardiocentesis** • **Beck's Triad**: **Hypotension**, **Muffled Heart Sounds**, **High JVP** (Distended neck veins).
Key 5	**Axis Deviation** ○ If QRS in lead I is **up** (+ve) and in lead II is **down** (negative) → **Left** axis deviation ○ If QRS in lead I is **down** (-ve) and in lead II is **up** (+ve) → **Right** axis deviation




-90°
aVF
-120°
II
-60°
III
-150°
aVR
EAD
LAD
-30
aVL
180°
-I
0°
+I
Normal
Range
RAD
+150°
aVL
+30°
aVR
+120°
III
+60°
II
+90°
aVF



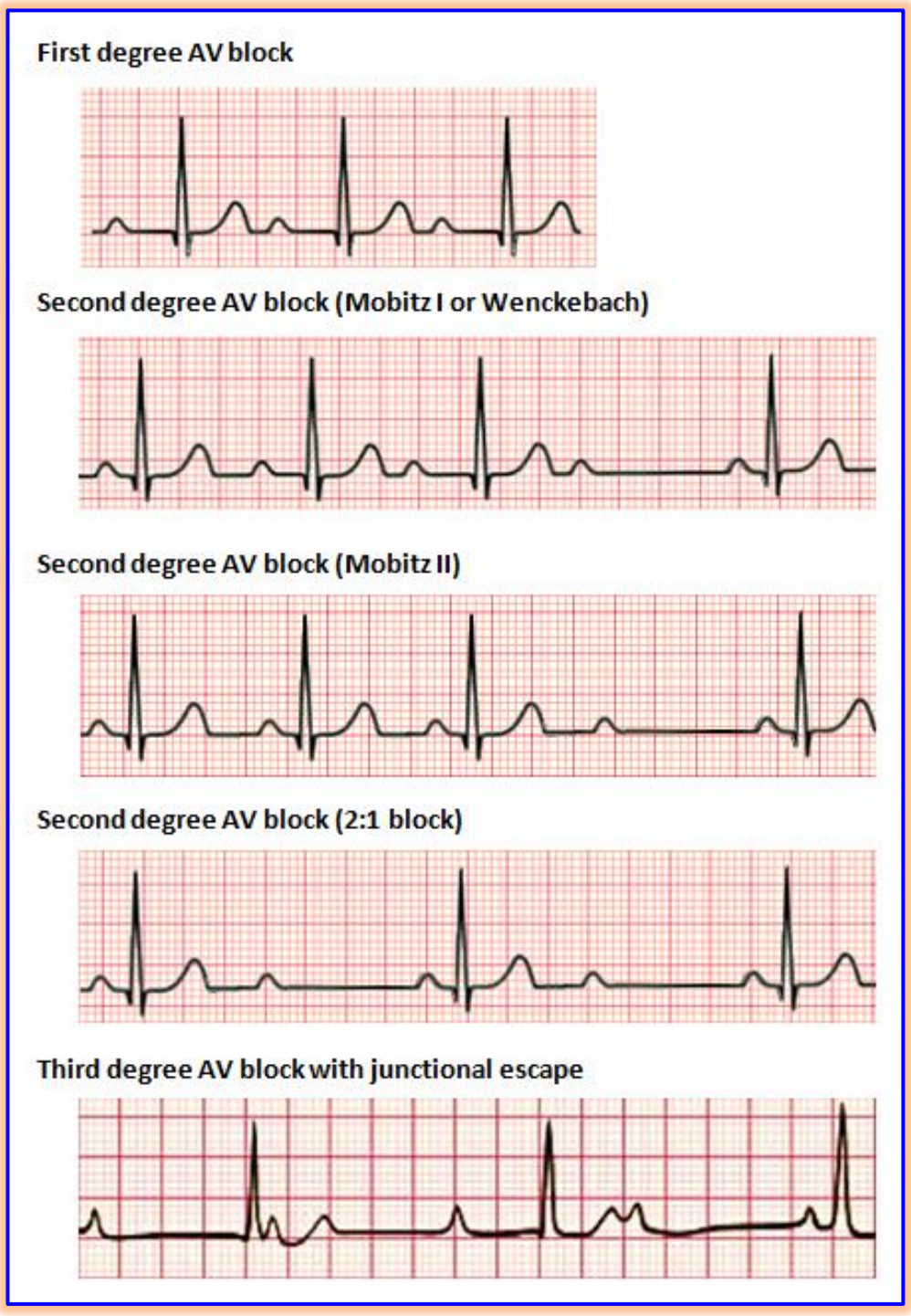
These causes are important!

Causes of Left Axis Deviation	Causes of Right Axis Deviation
Inferior MI	Lateral MI
Left Ventricular Hypertrophy	Right Ventricular Hypertrophy
Left Anterior Fascicular block (or hemiblock)	Left Posterior Fascicular Block (or hemiblock)
Obese	Thin, Tall, Children
Wolff Parkinson White Syndrome (delta wave)	Chronic Lung Disease
	Pulmonary Embolism

	• **Causes of <u>EXTREMER</u> Right Axis Deviation (No man's land) = (North west axis):** ○ **Congenital** Heart Disease. ○ Left Ventricular **Aneurysm**. For PLAB 1, you need to know either it is left axis deviation (Lead I is up and Lead II is down) or right axis deviation (Lead I is down and Lead II is up) and the causes of each (in the table above).
Key 6	**Types of heart block** **First degree heart block** • PR interval > 0.2 seconds (Only prolonged PR intervals). (i.e., PR interval occupies more than 1 large square (or 5 small squares). **Second degree heart block** • type 1 (Mobitz I = Wenckebach) → **Progressive prolongation** of the PR interval <u>until a dropped beat occurs</u>. • type 2 (Mobitz II) → PR interval is **constant** but the P wave is <u>often</u> not followed by a QRS complex.

Third degree (complete) heart block

- There is **no association** between P waves and QRS complexes.



Management of AV Heart Block:

◙ **1st Degree** Heart Block and **Mobitz type 1** usually

→ **do not require treatment** (as long as the patient is **Asymptomatic**).

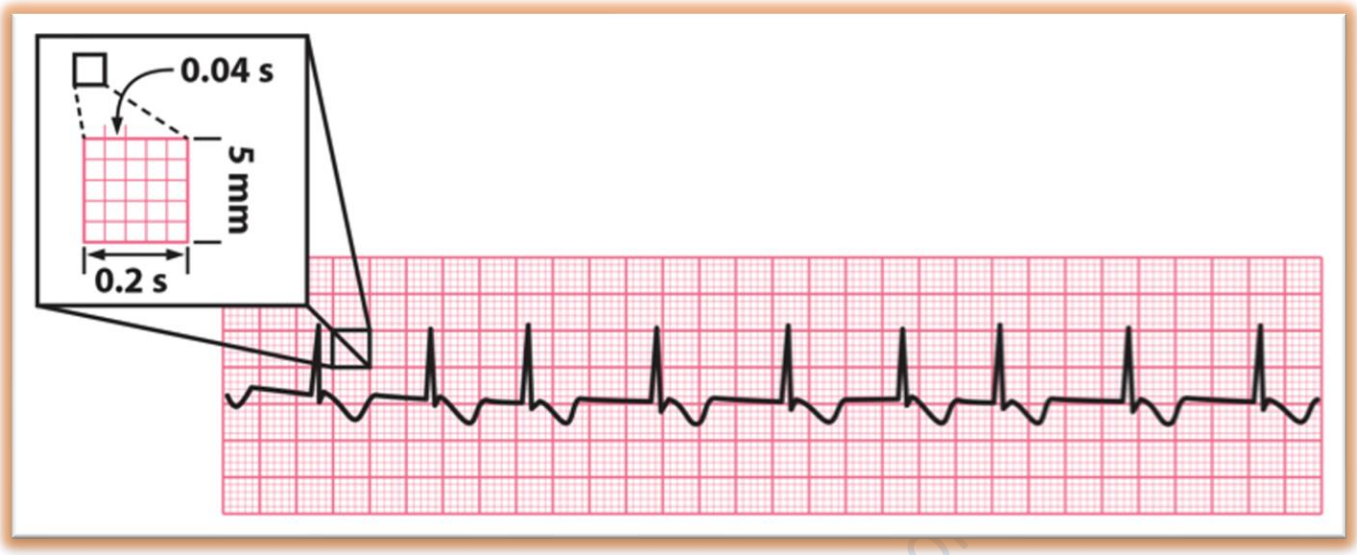
◙ **Mobitz type 2** and Complete heart block (**3rd degree** heart block)

→ require **permanent pacemaker**

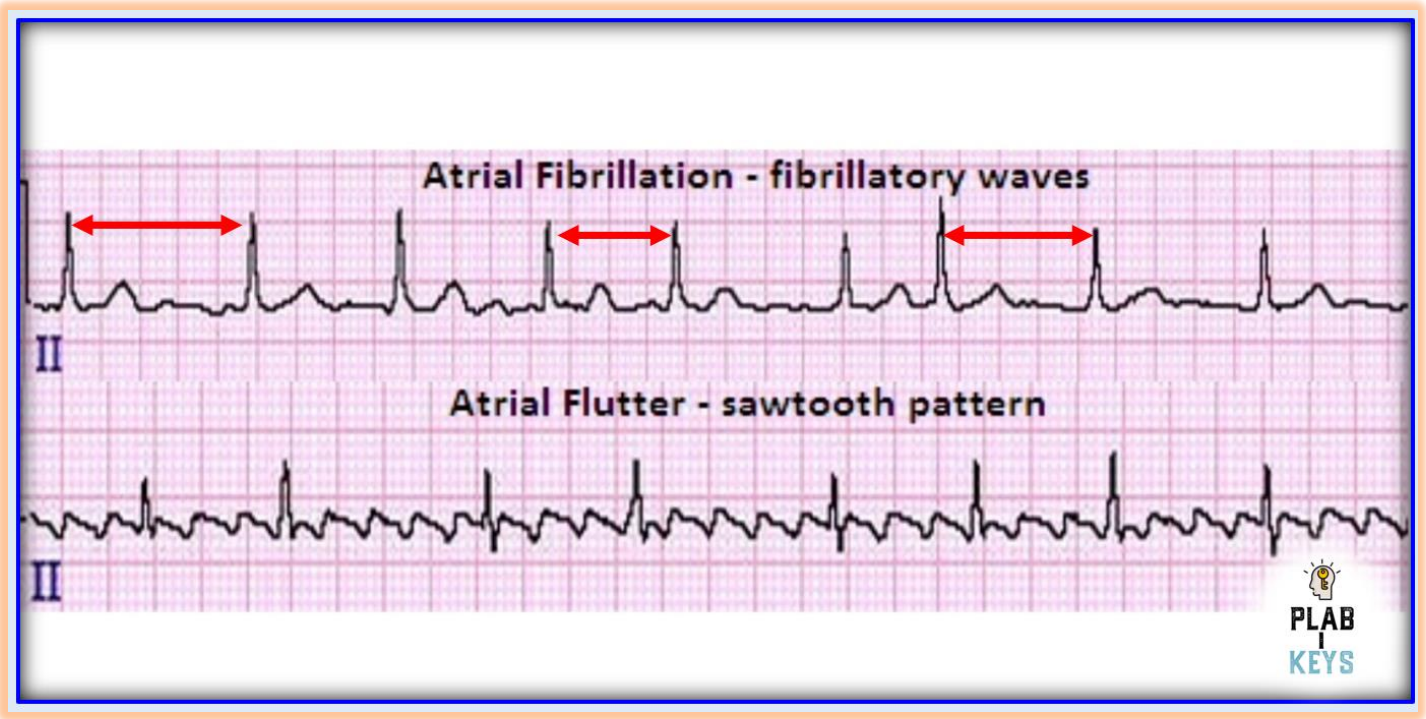
- **Initially → Atropine (first choice for all symptomatic bradycardia).**
- **Then → Transcutaneous pacing. (Used to buy time until transvenous pacing is done).**
- **Then → Transvenous pacing.**
- **Then → Permanent pacing (Pacemaker).**

For your knowledge:

- **1 small square = 0.04 sec.**
- **1 large square contains 5 small squares = 0.2 sec.**


0.04 s
5 mm
0.2 s

Key 7


Atrial Fibrillation - fibrillatory waves
II
Atrial Flutter - sawtooth pattern
II
PLAB
KEYS

- **Agents used to control rate (Rate Control) in patients with Atrial Fibrillation: (if the patient is stable and no hypotension)**
  - **Beta-blockers** (eg, atenolol, bisoprolol, metoprolol) → **First line** but Contraindicated in Asthma.
  - **Calcium channel blockers** **[non-dihydropyridine CCB]** (eg, diltiazem, verapamil) → used in **Asthmatic** patient.
  - **Digoxin** → (not considered first-line anymore as they are less effective at controlling the heart rate during exercise. However, they are the preferred choice if the patient has **coexistent heart failure**).

√ **If haemodynamically unstable** (eg, SBP ≤ 90) → **Cardioversion** (Shock).

√ If the patient with AF is **unstable** (eg, hypotension) and the AF has **just started** and no cardioversion in the options, Pick → **IV amiodarone**. **Imp √**

Note:

• Atrial fibrillation is a common arrhythmia after surgery. So, if it develops after surgery → Continue to observe and monitor vital signs.

• If it does not resolve on its own or if it worsens → Intervene (eg, bisoprolol).

- **Atrial Flutter** management → **Cardioversion** (**Shock**)

Key 8

Ventricular tachycardia

√ Ventricular tachycardia (VT) is broad-complex tachycardia originating from a ventricular **ectopic focus**.

√ It can develop into ventricular fibrillation and therefore requires urgent treatment.

√ P wave might be present or absent.

N.B:

- **ECG showing broad complex tachycardia in a (still) conscious patient even if semiconscious ± atrial activity and "haemodynamically STABLE"**

  → Ventricular tachycardia → Give Amiodarone He is stable!

- **ECG showing ventricular tachycardia in a haemodynamically unstable (e.g. SBP ≤ 90) patient → DC cardioversion = shock. He is unstable but has a pulse.**

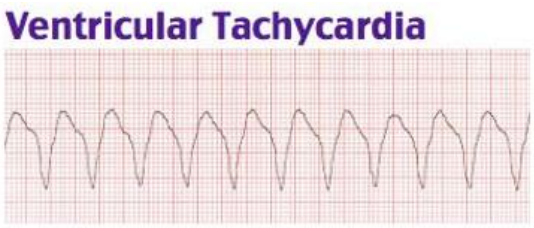
- **If the patient is Unconscious, Collapsed, or Not breathing + No Pulse!**

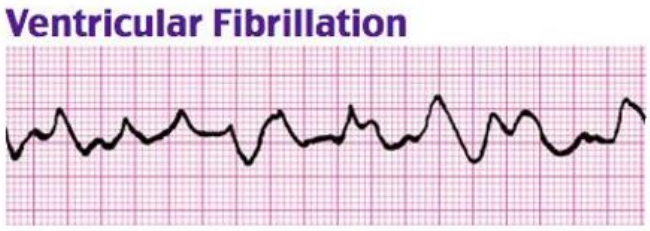
  → Ventricular Fibrillation → Defibrillation = Asynchronized shock

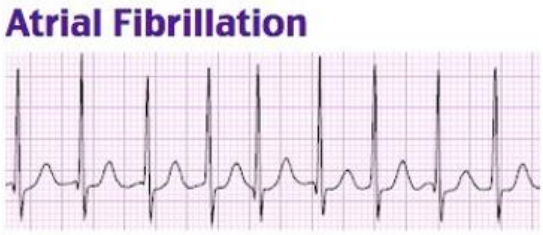
[As the patient is still conscious and with a felt pulse, it is likely ventricular tachycardia; not ventricular fibrillation. However, remember that ventricular tachycardia is managed by amiodarone if the patient is stable and by cardioversion if unstable]. If No pulse → Immediate Defibrillation.

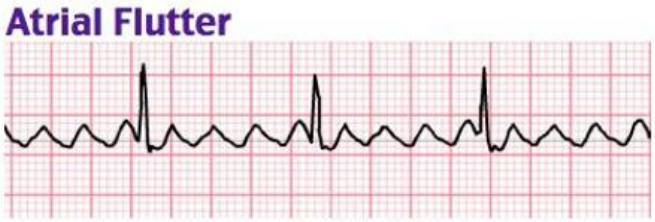
- **Ventricular fibrillation is the most important shockable arrhythmia.**

- **Hypokalemia (↓ K^+)** is the **most important cause of ventricular tachycardia (VT) clinically.**









Atrial Fibrillation

Palpitation, Tachycardia, Dyspnea, Fibrillatory waves on the ECG, Irregularly irregular rhythm

→ Give **Beta-Blocker**.

If Asthmatic → Give **Calcium Channel Blocker**.

If with hypotension → Cardioversion / IV amiodarone.

Atrial Flutter	''Fluttering Feeling in the chest'', Sawtooth waves on the ECG → **Synchronized ardioversion**.
Ventricular Tachycardia	Regular and Fast rhythm. Ongoing lightheadness, Palpitations, Chest pain. → Give **Amiodarone**. If unstable (SBP <90, ↓ consciousness) → **Immediate synchronized Cardioversion**. If Pulseless VT → **Immediate defibrillation**. Defibrillation = Unsynchronized cardioversion "high"
Ventricular Fibrillation	Older adult, Sudden collapse, Not breathing, Unconscious, **No pulse** → **''Immediate Defibrillation''**
Sinus Bradycardia	Lightheadness, hypotension, vertigo, syncope, dizziness. N.B. Sinus bradycardia is normal in young athletes. ◙ The first drug of choice for **Symptomatic Bradycardia** (Dizziness, feeling unwell) → **Atropine**
Sinus Tachycardia	Physiological situation (exercise, stress, anger).

	Hx of infection.
WPWS	Delta wave on the ECG
Narrow-complex Supraventricular tachycardia (SVT)	◙ Usually in **young** patients ◙ Presents with **Palpitations**, **Light-headedness**, **Recurrent**, Young ♠ Initial line → **Valsalva manoeuvre, Carotid massage**. ♠ Not improved? → **Intravenous adenosine**
Polymorphic (Broad-Complex) Ventricular Tachycardia **= Torsades De Pointes (TDP)**	◙ Beat-to-beat variations with **no uniform pattern** of ventricular contractions. ◙ **Broad QRS** (except in resting status), Prolonged QT, **Fainting** episodes, Patient might be a young athlete, Recurrent. ◙ Treatment → **IV Magnesium Sulphate**.

Key 9

Management of Congestive Heart Failure

*While **loop diuretics** (furosemide, bumetanide) and **nitrates** are important in the management of acute or decompensated cardiac failure, they have no effect on long-term survival.*

◙ The following medications have all been shown to reduce mortality in patients with left ventricular failure:

- ACE-inhibitors
- Beta-blockers
- Angiotensin receptor blockers (ARBs)
- Aldosterone antagonists (e.g. Eplerenone, Spironolactone)
- Hydralazine with nitrates

How to manage CHF? (Important)

- For all patient, for symptomatic relief and to reduce the volume overload

  → **Diuretics** (e.g., **Furosemide ▌Lasix ™**) or (**Bumetanide**).

- **Start** with either an **ACE inhibitor** or **Beta blocker** (one drug at a time).

- If the symptoms **persist** → **Add the other one** (ACEi or BB).

- If the symptoms **still persist** → **Add Spironolactone**

Side Notes:

√ Spironolactone is a potassium-sparing diuretic and an aldosterone antagonist.

√ (Important) If the patient of congestive heart failure is already on FUROSEMIDE, Beta blocker, and ACEi and still has lower limb edema → This means that the diuretic is not efficient (the furosemide) → Therefore, one of the following is needed:

- **Increase the dose of the furosemide.**
- **Switch the furosemide to either bumetanide or torsemide.**
- **Consider admission for IV loop diuretics.**

V. Imp. Note: If the patient has **Diabetes**, we start with **ACE inhibitors** (e.g. **Ramipril**) instead of Beta-Blockers.

ACE inhibitors are reno-protective and thus beneficial for diabetic patients.

"Try to link ACEi with DM in your mind"!

- **If HF + AF → Digoxin**

N.B. One might ask "Won't Furosemide + ACE inhibitors lead to hyperkalemia?

The answer is → No!

- Thiazide and Loop Diuretics (e.g. Furosemide) → HypOkalemia.
- ACEi (e.g., Ramipril) and Spironolactone → HypeRkalemia.

Key 10

The Summary of STEMI (ST-Elevation MI) Management

◙ **In Acute Settings**

→ **MONA**

(Morphine, **O**2, **N**itrates, **A**spirin 300 mg**)**

+ Heparin **(either unfractionated or LMW such as enoxaparin/ fondaparinux)**

	• If the patient presents **within 12 hours** of the onset of the symptoms → **PCI** (**P**ercutaneous **C**oronary **I**ntervention) **"The gold standard"** • **If Not, or PCI is unavailable** → Thrombolysis (**Alteplase**). **◙ (Chronic) Long-term Management of MI** Aspirin **for life,** Ticagrelor **or** Prasugrel **for 12 months** "Clopidogrel **previously",** Beta Blockers **(for 12 months),** ACE inhibitors**,** Statins **So, Long-term MI Rx = 5 Drugs: Aspirin, Clopidogrel, BB, ACEi, Statins** **AABC+S** → **A**spirin, **A**CE inhibitors, **B**eta-blockers, **C**lopidogrel + **S**tatins
Key 11	**Patent Foramen Ovale** √ The foramen allows blood to pass **from the right atrium** to **the left atrium**. √ The opening is supposed to close soon after birth, but sometimes it does not. In about **1 out of 4 people**, the opening never closes. If it does not close, it is called a PFO. √ In most of these individuals, the PFO causes no problems and remains undetected throughout life.

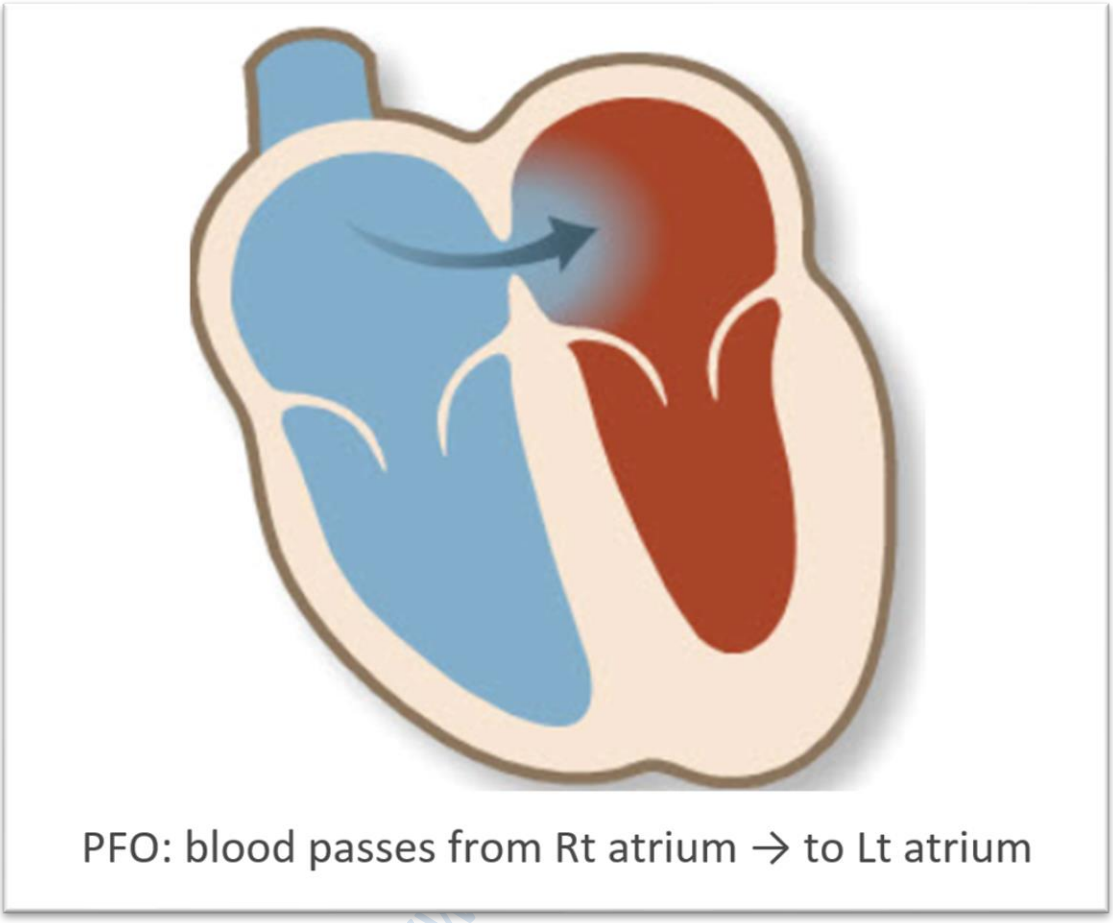
√ PFO has long been studied because of its role in paradoxical embolism (an embolism that travels from the venous side to the arterial side). This may lead to a stroke or transient ischemic attack.

√ **Transesophageal echocardiography** is considered the most accurate investigation to demonstrate a patent foramen ovale.

√ A patent foramen ovale may also be an incidental finding.

The important point to remember is:

Trans-oesophageal Echocardiography (TOE) with bubble contrast is the gold standard in diagnosing Patent Foramen Ovale (PFO).



Key 12

Important Complications of Myocardial Infarction (MI)

Cardiac arrest

- This most commonly occurs due to patients developing **ventricular fibrillation** and is the most common cause of death following a MI.
- Patients are managed as per the ALS protocol with **defibrillation.**

Chronic heart failure

If the patient survives the acute phase, their ventricular myocardium may become dysfunctional resulting in chronic heart failure.

Management:

◙ For all patient for symptomatic relief and to reduce the volume overload → **Loop Diuretics** (e.g., **Furosemide**)

◙ **Start** with either **ACEi** or **BB**. (One drug at a time)

(Remember that ACEi and ARBs is delt with as similar in effect).

◙ If the symptoms **persist** → **Add the other one** (ACEi or BB).

◙ If the symptoms **still persist** → **Add Spironolactone** (Aldosterone Antagonist).

[**Important**: If a patient is already on an **ACE inhibitor** (eg, lisinopril) or is already on **ARBs** (eg, losartan, candesartan) and he needed an additional medication for his chronic heart failure, pick a **beta-blocker** eg, bisoprolol].

If he is already on an ACEi or ARBs + BB → go for spironolactone.

Tachyarrhythmias

◙ **Ventricular fibrillation**, as mentioned above, is the **most common cause of death following a MI.** Other arrhythmias can also occur e.g. ventricular tachycardia.

◙ Management:

1) Check the patient's pulse, if no pulse, commence the arrest protocol immediately (and deliver immediate defibrillation)

2) Administer O2.

Pericarditis "important √"

◙ Occurs within **48 hours (i.e. 2 days)** after MI.

◙ **Features → Pleuritic chest pain that is worse on lying flat and during inspiration ± Fever ± Pericardial rub**.

◙ Pericardial effusion may develop leading to enlarged globular heart on chest X-ray and is confirmed by echocardiogram.

◙ **ECG → Widespread Saddle Shaped ST Elevation** with **upward concavity** + PR Depression.

◙ Management of Pericarditis (imp):

√ A full-dose **NSAID** should be used (eg, aspirin, 2-4 g/d; **ibuprofen** 1200-1800 mg/d; indomethacin 75-150 mg/d); treatment should last at least 7-14 days.

√ **Colchicine** (as an adjunct to NSAIDs to ↓ inflammation and recurrence).

Dressler's syndrome "important √"

◙ Similar to pericarditis in features but it tends to occur **2-6 weeks** following a myocardial infarction.

◙ The underlying pathophysiology is thought to be an *autoimmune reaction* against antigenic proteins formed as the myocardium recovers.

◙ It is characterised by a combination of **fever, pleuritic chest pain that worsens on inspiration and lying flat, pericardial effusion and a raised ESR**.

◙ It is treated with **NSAIDs**.

◙ **ECG: Widespread Saddle Shaped ST Elevation ± PR Depression.**

Left ventricular aneurysm

◙ The ischaemic damage sustained during a MI episode may weaken the myocardium resulting in a thin muscular layer; thus, aneurysm formation.

◙ This usually **occurs 4-6 weeks post MI**.

◙ This is typically associated **with persistent ST elevation and left ventricular failure**.

◙ ***A thrombus*** *may form within the aneurysm increasing the risk of* ***stroke****. Patients are therefore* ***anticoagulated****.*

ECG → Persistent ST Elevation *+ Left Ventricular Failure.*

CXR → *Enlarged heart with a **bulge** at the left heart border.*

Echo → *Paradoxical movement of the ventricular wall.*

Ventricular septal defect (VSD)

◙ Rupture of the interventricular septum usually occurs in the ***first week*** after a MI attack and is seen in around 1-2% of patients.

◙ Features: acute heart failure associated with **a pan-systolic murmur** best heard at the left lower sternal border + Heart Failure (eg, bibasilar crackles) + Shock (hypotension, tachycardia).

◙ An **echocardiogram** is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion.

◙ **Urgent surgical correction** is needed.

Acute mitral regurgitation (MR): "important √" pansystolic murmur

◙ Occurs **2-15 days** after the MI (Mostly **inferior** MI).

◙ Due to → Ischemia or **rupture of the papillary muscles** of the mitral valve.

◙ An early-to-mid systolic or **Pansystolic murmur** is typically heard at **Apex**.

◙ May present with Hypotension, Tachycardia, and Pulmonary edema.

If with Pulmonary Edema → SOB (Dyspnea), Bi-basal Crackles, Tachycardia.

◙ To **diagnose** MR → **Echocardiogram**.

◙ **Treatment** → vasodilators, but often requires emergency surgical repair.

Acute Tricuspid regurgitation (MR):

Similar to mitral regurgitation but the **pan-systolic murmur** is heard over the **lower left sternal border**.

Important Note:

◙ **Pericarditis** (Can occur as a Complication of MI, may develop shortly after MI within **2 days**) and **Dressler's syndrome** (presents **2-6 weeks** after MI) both have the same features:

→ **Pleuritic chest pain that worsens on lying flat and during inspiration**, and improves on upright sitting forward.

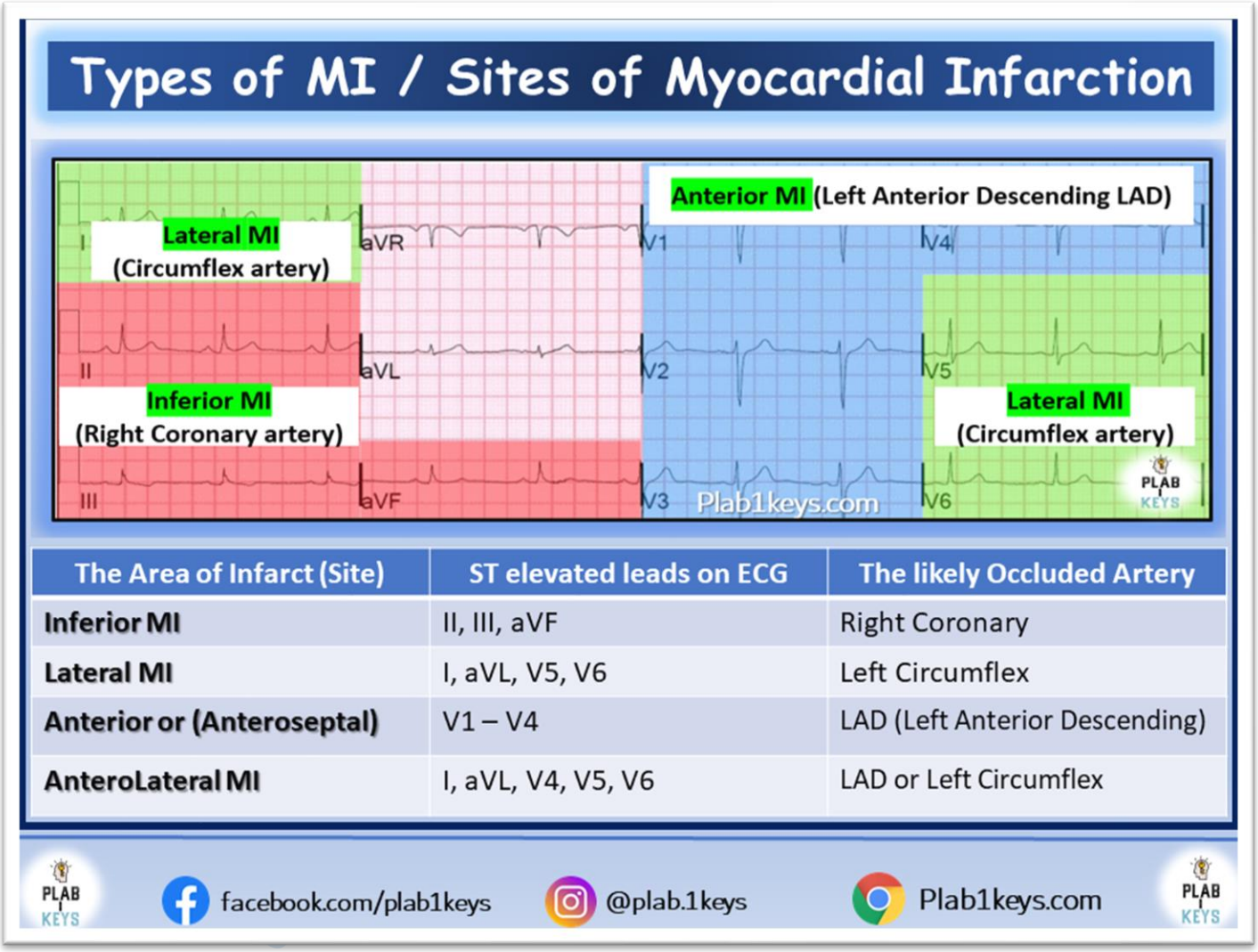
± **Pericardial rub**,

± **Widespread Saddle shaped ST elevation** on the ECG.

◙ They can also lead to **Pericardial effusion** (**Enlarged globular heart** on chest X-ray) and if severe enough, **Cardiac Tamponade** can also develop (also **enlarged globular heart** of the X-ray + Beck's Triad: Hypotension, Muffled

Heart Sounds, High JVP).

Key 13



The Area of Infarct (Site)	ST elevated leads on ECG	The likely Occluded Artery
Inferior MI	II, III, aVF	Right Coronary
Lateral MI	I, aVL, V5, V6	Left Circumflex
Anterior or (Anteroseptal)	V1 – V4	LAD (Left Anterior Descending)
AnteroLateral MI	I, aVL, V4, V5, V6	LAD or Left Circumflex

Wide spread ST depression (+) **ST elevation in aVR**

→ Left main coronary artery [LMCA] occlusion

→ Emergency coronary angiography

Key 14	**For Theoretical Exams:** ◙ **Any patient presents with STEMI:** → Give **MONA** (**M**orphine, **O**2, **N**itroglycerin, **A**spirin) and then: → Send immediately for **PCI** (Percutaneous Coronary Intervention). • If PCI is not obtainable → **Alteplase**. [i.e. thrombolysis]. • If PCI and Alteplase are not given, pick →**Streptokinase**. [i.e. thrombolysis]. --- ◙ **Any patient presents with NSTEMI or Unstable angina**: → **After giving morphine, 2 medications should be given immediately**: √ **Oral Aspirin 300 mg** (+) √ SC **Low molecular weight heparin** "or" SC **Fondaparinux**. --- • If **left main coronary artery occlusion:** i.e. (widespread ST depression + ST elevation in aVR): after giving morphine → **Emergency coronary angiography**.
Key 15	◙ The **first drug of choice** for **Symptomatic Bradycardia** (Dizziness, feeling unwell) is → **Atropine**

	(Given 0.5 mg IV push and may be repeated up to a total dose of 3 mg). **What if he was given atropine but no response?** **Next step would be → Temporary transcutaneous pacemaker.** ◙ 2nd Line → Dopamine. ◙ 3rd Line → Epinephrine. ♠ N.B. If the question was "***the next best step***" (**or**) "***the initial line***", the Answer will be → **O2 (ABCD)**.
Key 16	**Beck's Triad**: **√ Hypotension**, **√ Muffled "faint = weak" Heart Sounds**, **√ High Jugular Venous Pressure [JVP]** (= **Distended neck veins**). → **Cardiac Tamponade**. → **Echo for Dx** and **Pericardiocentesis for Rx**
Key 17	**Infective Endocarditis (IE)** **New Murmur** + **Fever** → think of **Infective Endocarditis (IE)** ± Malaise, Rigors.

The initial step → **Blood Culture** √ Then → **Echo**

Example:

6 weeks following MI, a 59-year-old patient presented with a 10-day history of intermittent fever and a new-onset of a soft high-pitched murmur at the apex. His ECG is normal.

Fever + **New-onset murmur**, Think → **Endocarditis**.

- **Risk Factors:**

◙ **A previous episode of endocarditis** → the **strongest risk factor.**

◙ **Rheumatic valve disease.**

◙ **Prosthetic valves.**

◙ Congenital heart defects.

◙ **Intravenous drug users** (IVDUs: typically causing tricuspid lesion).

- **The Causative Organisms:**

- **Staph. Aureus** is the commonest cause of IE in general.

- **Staph**. **Epidermidis** is the commonest cause after prosthetic valve surgery.

- **Strept**. **Viridans** (especially sterpt. Mitis and strept. Sanguinis) are the commonest cause in people with poor dental hygiene or following a dental procedure.

• Features and Diagnosis → Modified Duke criteria

Infective endocarditis is diagnosed in any of the following situations:

- 2 major criteria, or
- 1 major and 3 minor criteria, or
- 5 minor criteria

Major criteria

1) Positive blood cultures

◙ Two positive blood cultures showing typical organisms consistent with infective endocarditis, such as Streptococcus viridans and the HACEK group, (**Or**)

◙ Persistent bacteraemia from two blood cultures taken > 12 hours apart or three or more positive blood cultures where the pathogen is less specific such as Staph aureus and Staph epidermidis.

Not to be confused, it is true that staph. Aureus is the commonest pathogen in IE; however, it is not specific for IE as it causes many other inflammations.

2) Evidence of endocardial involvement (i.e. +ve Echo for IE)

◙ Positive echocardiogram (oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves). (**Or**)

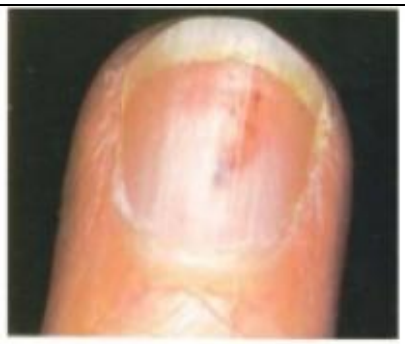
◙ New valvular regurgitation

Minor criteria

1. Predisposing heart condition or **intravenous drug use**.
2. Microbiological evidence that does not meet the major criteria.
3. Fever > 38°C.
4. **Vascular phenomena** → Major emboli, Splenomegaly, Clubbing, Splinter haemorrhages, Janeway lesions, Petechiae or purpura.
5. **Immunological phenomena** → Glomerulonephritis, Osler's nodes, Roth spots.

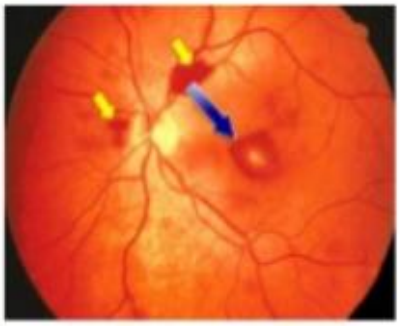
N.B.

- **Osler's Nodes**: painful, red nodules on the hands or feet that can persist for hours to days.
- **Janeway lesions**: Non-tender, small, erythematous or hemorrhagic macular or nodular lesions on the soles or palms. (they occur due to septic micro-emboli that deposit the bacteria under the skin).



Splinter hemorrhage

Osler node



Roth's spot

Janeway lesion

Endocarditis: initial "Empirical" "Blind" therapy

- **Native valve endocarditis** →

- Amoxicillin + low-dose Gentamicin. (Or),
- Vancomycin + low-dose Gentamicin (If Penicillin allergic or MRSA "Methicillin-Resistant Staph. Aureus" is suspected or Severe Sepsis).

- **If Hx of prosthetic valve endocarditis**→

Vancomycin + low-dose Gentamicin + Rifampicin

The most important note to remember is that in any patient presenting with **Fever** + a new heart **Murmur** → suspect **Infective Endocarditis** and order **Blood Culture** until proven otherwise.

Example 1:

A man who had dental extraction a few days ago presents with petechia. His vitals are stable except his body temperature which is 38.9. On examination (O/E): He has petechiae, painful nodules on his palms, and a cardiac murmur.

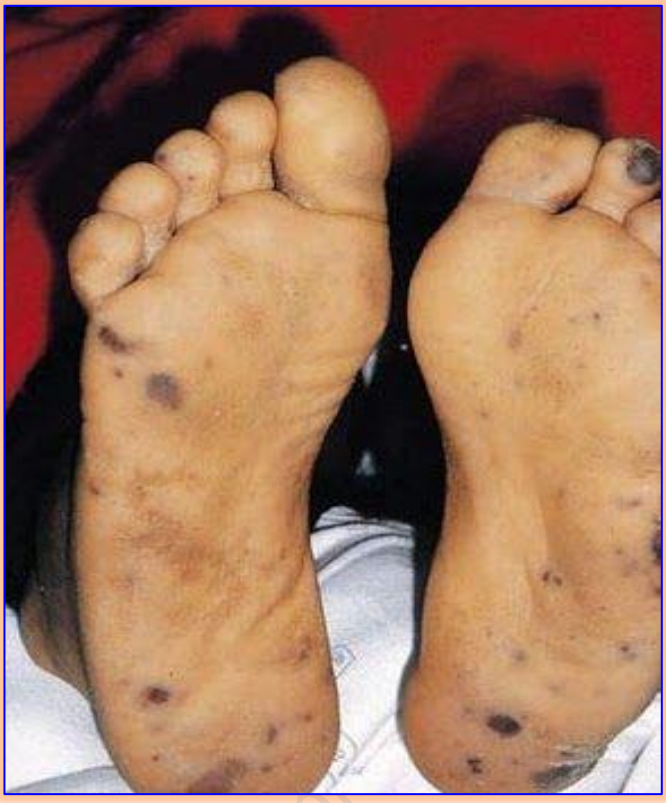
The likely Dx → **Infective Endocarditis**. (Fever + New Murmur).

The underlying cause of this condition → **infection**.

The next investigating step → **blood culture** (followed by Echo).

Example 2:

A man presents with Fever, confusion, petechiae. This is a picture of his soles



What is the most appropriate investigation?

→ **Blood Culture**

These lesions are likely **Janeway lesions** (minor criteria of infective endocarditis).

♦ Likely → **Infective endocarditis**

→ Do Blood culture **then** Echocardiogram

Key 18

◙ **In a patient with Atrial Fibrillation → We use the CHA2DS2-VASc Score**

"To determine the need to anticoagulants".

	Condition	Points
C	**Congestive heart failure** (or LV dysfunction)	1
H	**Hypertension** BP>140/90 or treated hypertension on medication	1
A_2	**Age** ≥ 75 years	2
D	**Diabetes Mellitus**	1
S_2	**Prior Stroke** or **TIA** or **Thromboembolism**	2
V	**Vascular disease** (e.g. MI, PVD, Aortic plaque)	1
A	**Age 65-74 years**	1
S_c	**Sex category** (female gender)	1

◙ Give **Warfarin** or **DOAC** (**D**irect-Acting **O**ral **A**nti**C**oagulants, such as **Apixaban**, **Rivaroxaban**, **Edoxaban, Dabigatran**) To:

√ **All** patients with score **≥ 2**.

√ Consider giving Warfarin or DOAC to **Men** who score **≥ 1**.

Advantages of DOAC:

- No need for INR Monitoring,
- Faster Onset of Action (2-4 hours),

- Reduces the risk of intracranial Hemorrhage.

Disadvantages of DOAC:

- No Antidote
- Require strict compliance by the patients.

Important Scoring Systems to Know

◙ The **CHA2DS2-VASc score** is used to determine **the need to anticoagulants** in a patient who has **atrial fibrillation**. **√ important**

◙ The **ABCD2 score** (Prognostic) is used to **identify the risk of future stroke in patients who have had a suspected TIA in the following 7 days. √ Not advised to be used now according to the recent 2019 CKS guidelines.**

◙ The **HAS-BLED score** estimates the **risk of major bleeding** for patients **on anticoagulation** for **atrial fibrillation.**

◙ The **DRAGON score** predicts **the 3-month outcome in ischaemic stroke patients receiving tissue plasminogen activator (tPA) e.g. alteplase.**

	◙ The **QRISK2 score** is used to **determine the risk of a cardiovascular event in the next 10 years**.
Key 19	**Pulmonary edema** **Mechanism:** Often caused by **congestive heart failure**. When the **heart** is not able to pump efficiently → blood may return into the veins → then to the lungs. As the pressure in these blood vessels increases, fluid is pushed into the air spaces (alveoli) in the lungs. **Features:** **Desaturation** (Low O2 Sat.), **Dyspnea** (SOB), **Orthopnea** (SOB worsens when lying down), Auscultation → **Crepitations** "**Crackles** = **rales**". Mostly **bi-basal**. **Tachycardia**. **Investigations:** While **chest X-ray** usually shows features of pulmonary edema **(The single most appropriate Investigation)**, the underlying cause requires

Echocardiogram to be identified (e.g. Congestive Heart Failure, Complication of MI → Acute Mitral Regurgitation due to papillary rupture, Ventricular aneurysm, …etc.)

Therefore, pay attention to the question words!

◙ The Most Appropriate Investigation → **Chest X-Ray**. "imp √"

◙ The Investigation Needed to Identify the Underlying **Cause** → **Echo**. "imp √"

Management:

MONA (But the last **A** -**A**spirin- is replaced by **F** -**F**urosemide-):

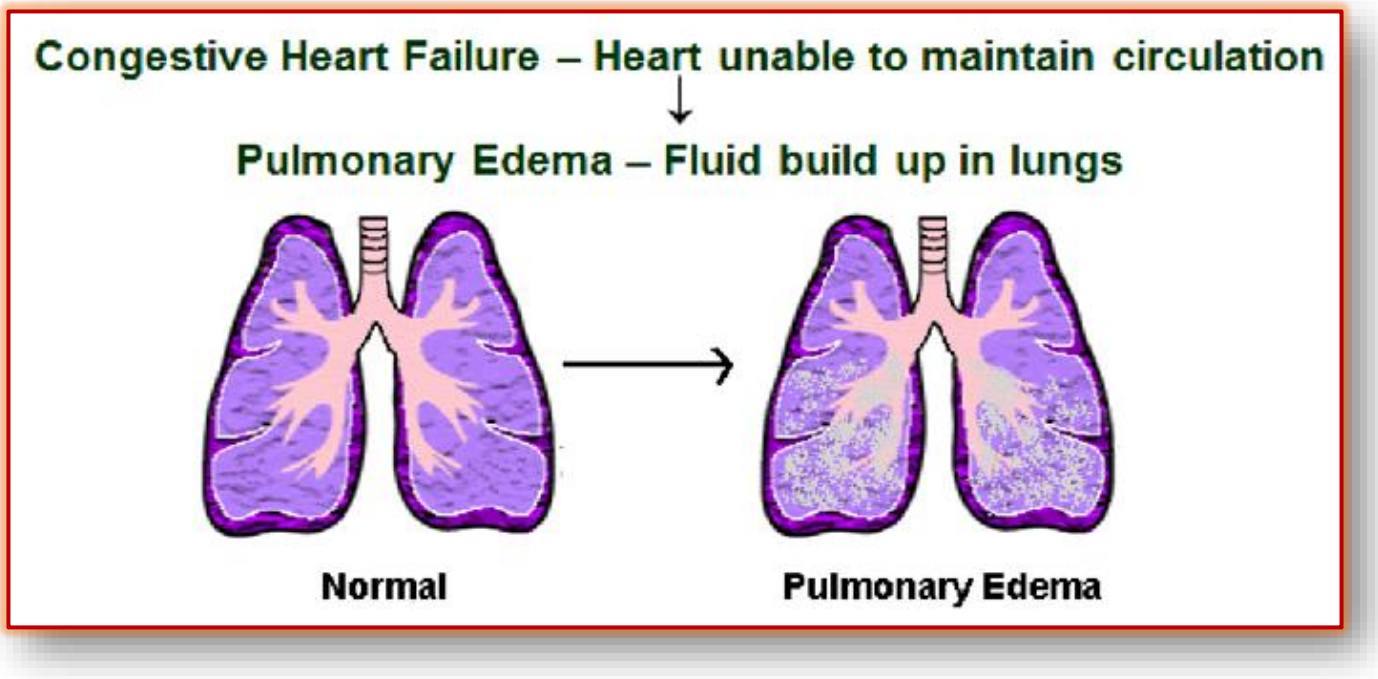
→ **Morphine**, **O2**, **Nitrates**, **Furosemide** (Lasix).

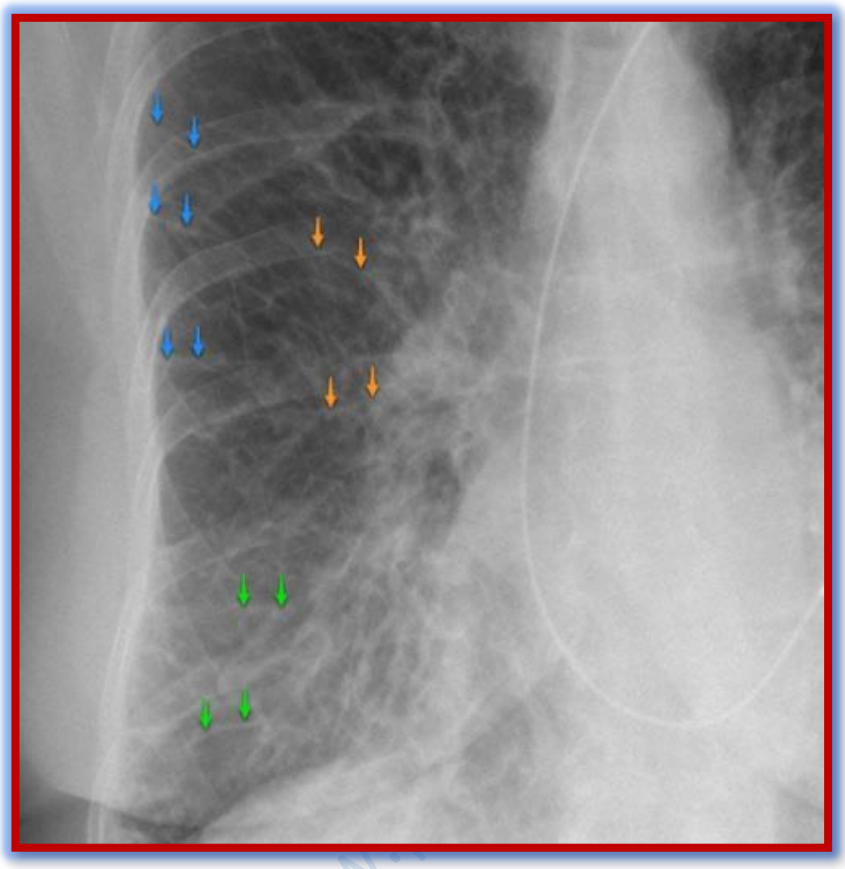
1) Sit the patient up (Popup position) and give **O2** (aim for O2 saturation of ≥ 95%, or ≥ 90% in COPD patients).
2) Spray 2 puffs of sublingual **GTN** (Glyceryl TriNitrates).
3) Give **Furosemide** (Lasix) 40 mg IV (Slowly).
4) Give **Diamorphine** (2.5-5 mg IV slowly) **or Morphine** (5-10 mg IV slowly) to relieve pain, anxiety and distress.

N.B. A good difference between **Pulmonary Edema** and **Pulmonary Embolism** is that Pulmonary Oedema can be diagnosed by Chest X-ray

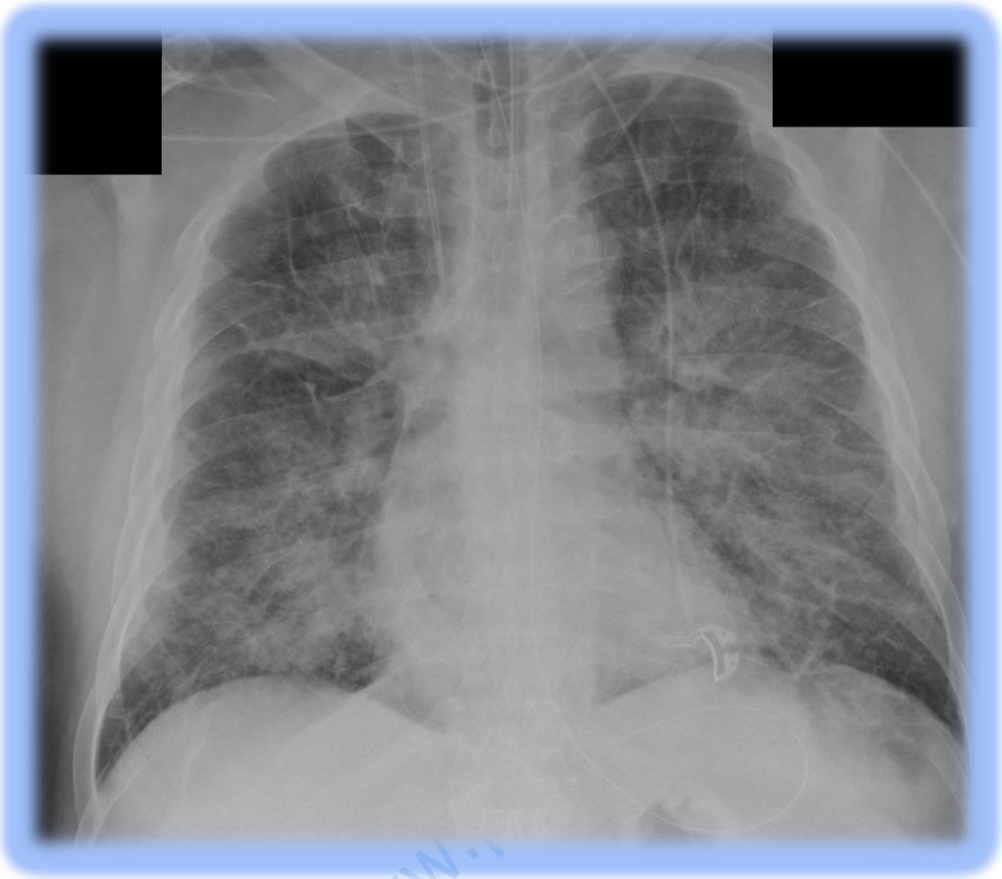
while Pulmonary Embolism needs CTPA (CT Pulmonary Angiogram).

This might be given as a hint in a question.





Pulmonary Oedema → Kerley Lines (Expansion of the interstitial space by fluid)



Pulmonary Oedema → Kerley Lines + Bat's wing hilar shadow

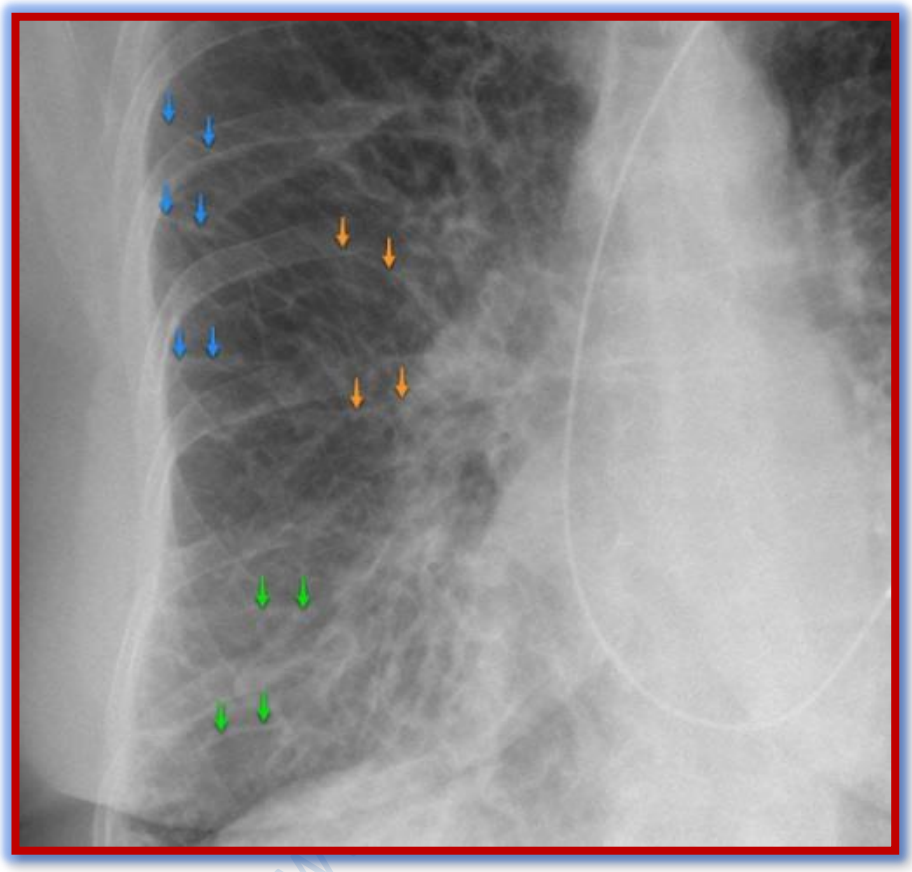
Key 20

Scenario

20 days after MI, a patient developed sudden Dyspnea. O/E → Tachycardia, Desaturation (88% on Room Air), Hypotension and Bilateral Chest Crackles.

- **The likely Dx → Pulmonary Oedema.**
- **The appropriate Initial investigation → Chest X-Ray.**
- **The best investigation to identify the cause → Echocardiography.**

◙ **Treatment** → **MONF** (**Morphine**, **O2**, **Glyceryl Trinitrates**, **FUROSEMIDE**).



Pulmonary Oedema → **Kerley Lines** (**Expansion of the interstitial space by fluid**)

Important:

If pulmonary edema is cuased by existent hear failure, the patient needs to be discharged on either ACE inhibitor or a beta blocker (one drug at a time).

If asthmatic → ACE inhibitor is preferred over BB as BB may worsen asthma.

Key 21

The Typical Presentation of Acute MI (75% of cases):

◙ Central Chest Pain or Epigastric pain or Substernal pain that is severe, sudden, crushing, pressuring, squeezing, constricting or burning.

◙ Radiates to arms, shoulders, neck or jaw.

◙ ± Sweating (Diaphoresis), Nausea, Vomiting, Fatigue and/or Palpitations.

◙ SOB "Shortness of breath".

Important DD: [Dissecting Aneurysm] Aortic dissection

Although Dissecting aortic Aneurysm may have more or less a similar presentation to MI, to be chosen as an answer, there should be other clinchers pointing towards dissecting aneurysm such as:

√ **Unequal pulses in upper limbs**.

√ **Hx of Marfan Syndrome (tall, long slender limbs and fingers).**

√ **Hx of Ehlers-Danlos syndrome/ turner syndrome**.

√ **Severe tearing chest pain that radiates to the Back**.

√ **HTN is the most important risk factor**.

√ **The patient presents with Hypotension, SOB, tachycardia, sweating**.

Points on Aortic Dissection

• Aortic dissection is a rare but serious cause of chest pain.

• Pathophysiology → tear in the tunica intima of the wall of the aorta.

• Injury of the innermost layer of the aorta allows blood to flow between the layers of the aortic wall, forcing the layers apart.

• In most cases, this is associated with a sudden onset of severe chest or back pain, often described as "tearing" in character. Also, vomiting, sweating, and lightheadedness may occur.

• Other symptoms may result from decreased blood supply to other organs, such as stroke or mesenteric ischemia.

• Aortic dissection can quickly lead to death from not enough blood flow to the heart or complete rupture of the aorta.

• The transoesophageal echocardiogram (TEE) is a good test in the diagnosis of aortic dissection, with a sensitivity up to 98% and a specificity up to 97%. It has become the preferred imaging modality for suspected aortic dissection.

• Other good investigations → CT scan with contras/ MRI.

• In emergency settings → US or CT scan.

• **Stanford classification of Aortic Dissection:**

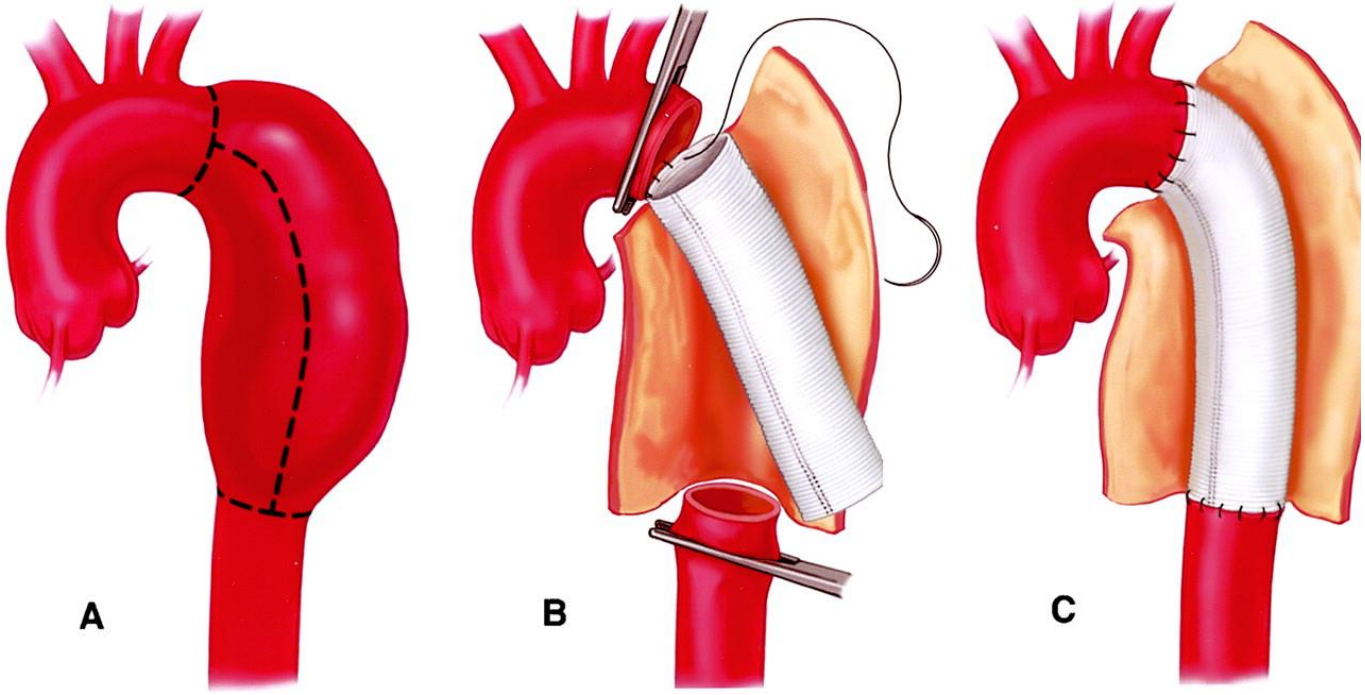
type A: ascending aorta, 2/3 of cases

type B: descending aorta, distal to left subclavian origin, 1/3 of cases

◙ Management od Aortic Dissection

• Type A → surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention.

• Type B → conservative management, bed rest, reduce blood pressure: IV labetalol to prevent progression



Key 22

Left Bundle Branch Block (LBBB)

In the context of chest pain, new LBBB is significant as it is an indication for **thrombolysis / percutaneous coronary intervention (PCI)**.

LBBB features on ECG:

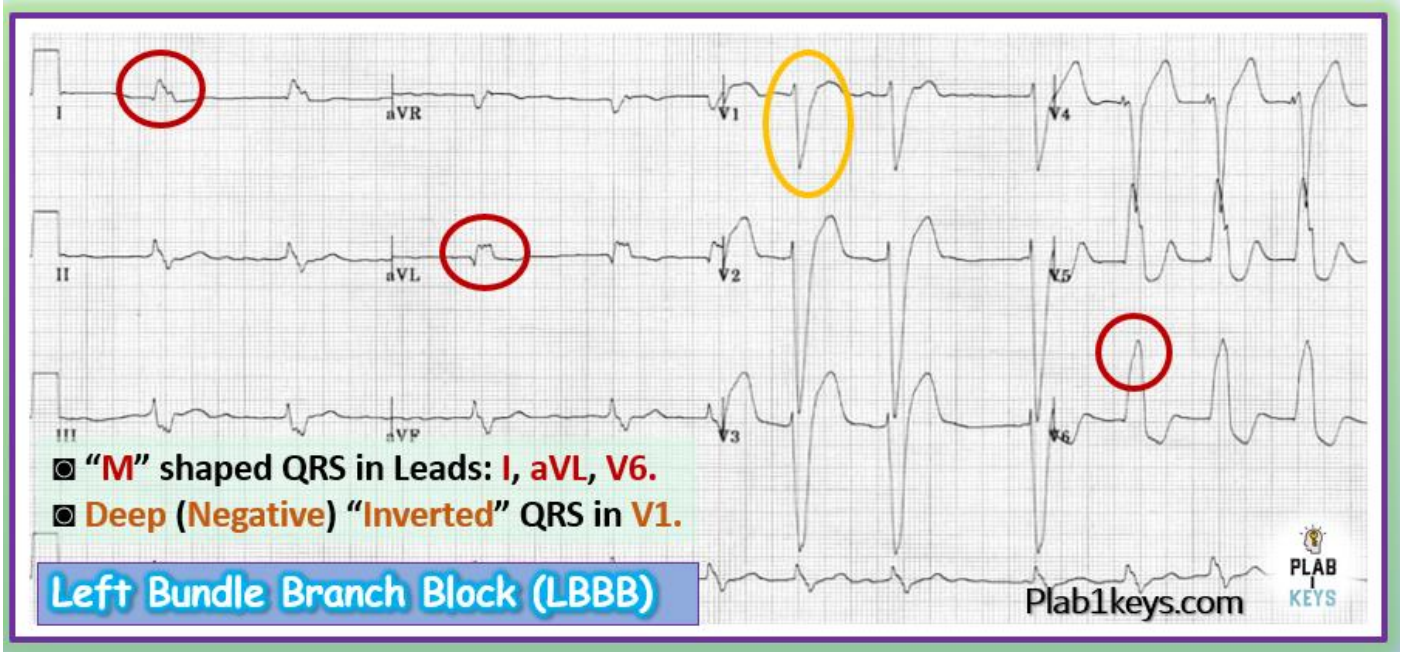
◙ Notched (**M shaped**) broad complex QRS: usually in Lead (**I**), **aVL** and **V6** but not always.

◙ Deep inverted (**Negative**) QRS: usually in lead (**V1**).

◙ Left Axis Deviation (Not always)

Important Note

⇒ **A new onset LBBB is characteristic for Myocardial Infarction (MI)**



Key 23

Ruptured Abdominal Aortic Aneurysm (AAA)

◙ **The classical picture**: a triad of:

Pain, **Hypotension**, **pulsatile tender abdominal mass**.

- **Sudden onset** severe abdominal ± Lower back ± Flank pain.
- **Shock** (Hypotension, Sweating, Fainting)
- **Absent Lower Limb Pulse, mottled skin.**

◙ It is a **surgical emergency**; therefore, immediate **Ultrasound** is the most appropriate initial investigation.

◙ If no U/S in the options, go for **CT scan** abdomen.

◙ **Screening for Abdominal Aortic Aneurysm (AAA) in the UK:**

√ **Men only.**

√ **Once only.**

√ **In 65th year.**

√ **by Ultrasound.**

Key 24

Management of Chronic Heart Failure

In a patient with **Heart Failure**

[**LL Edema**, **Dyspnea**, **Orthopnea**, **Ejection fraction less than 40%**],

The management would be:

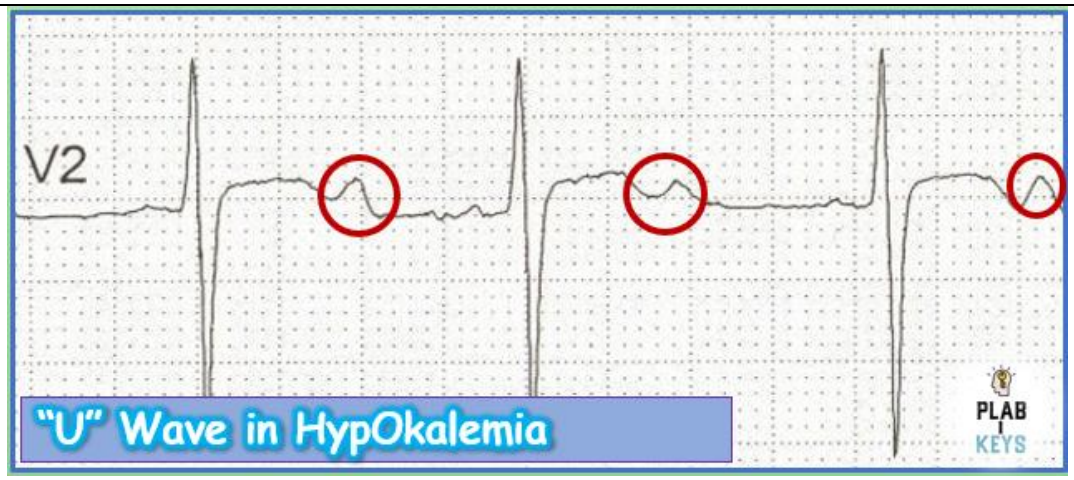
- For symptomatic relief and to reduce the volume overload, all patients should receive → **Diuretics** (e.g. loop diuretics e.g. **Furosemide**)
- **Start** with either **ACEi** or **BB**. (one drug at a time)
- If the symptoms **persist** → **Add the other one** (ACEi or BB).
- If still symptomatic → **Add spironolactone** "potassium sparing diuretics".

- **V. Imp. Note**: If the patient with **Heart Failure** has **Diabetes**, we start with **ACE inhibitors** (e.g. **Ramipril**) instead of Beta-Blockers.

"Try to link **ACEi** with **DM** in your mind"!

	Even in hypertension "as you will see in the coming keys", any patient despite the age and ethnicity who has diabetes and HTN, start with ACE inhibitors as step 1.
Key 25	**Coronary Artery Dominance** • The artery that supplies the **Posterior Descending Artery** (**PDA**) determines the coronary dominance. • In **85%** of the population, the **Right coronary artery (RCA)** gives off the PDA (Right Dominant). • In **15%** of the population, the **left circumflex** gives off the PDA (Left Dominant). ◙ Hence, the artery that has artery dominance is the (**RCA**), as it gives off the PDA in **85**% of people.
Key 26	**Dressler's syndrome** ◙ It tends to occur around **2-6 weeks** following a MI. ◙ The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers.

	◙ It is characterised by a combination of **fever**, **pleuritic pain worsens on inspiration** and **on lying flat**, **pericardial effusion** and a **raised ESR**. ◙ **ECG** → Widespread Saddle Shaped ST Elevation + PR Depression ◙ It is treated by **NSAIDs**. ◙ Its features are more or less similar to acute pericarditis. However, pericarditis usually occurs only a few days after MI.
Key 27	**Hypokalemia** ◙ **Muscle weakness and cramps** + **U wave on the ECG** ◙ One important reason for hypokalemia is **Thiazide like Diuretics** (e.g. **Bendroflumethiazide**) and **Loop diuretics** (e.g. **Furosemide**) But not Potassium-sparing diuretics (e.g. Spironolactone) which causes HypeRkalemia. ◙ **Spironolactone**, **ACE inhibitors, ARBs** → **HypeRkalemia**. ◙ **Loop diuretics**, **Thiazide like diuretics** → **HypOkalemia**. ◙ The ECG changes in Hyp**U**kalemia → **U** Wave



U wave (hypokalemia) → an additional wave after (T-wave)

Management of hypokalemia

1) Oral or IV **Potassium chloride** (based on severity), e.g. if K+ <2.5 → IV.
2) Stop/ Treat the cause (e.g. stop furosemide, thiazide like diuretics).

HypOkalemia	HypeRkalemia
• **Loop Diuretics** (e.g. Furosemide) • **Thiazide-like diuretics** (e.g. bendroflumethiazide, indapamide) • **Vomiting** and **Diarrhea** • **Villous Adenoma** • **Renal tubular failure** • **Cushing Syndrome** • **Conn's disease** (1ry hyperaldosteronism)	• **ACE inhibitors** (e.g. enalapril). • **ARBs** (e.g. losartan). • **Potassium-sparing diuretics** (e.g. Spironolactone/ Eplerenone) • **CKD/ Acute renal failure** • **Addison's** (1ry Adrenal Insufficiency). • **Congenital Adrenal Hyperplasia (CAH)**.

	Remember: √ Severe vomiting can cause hypokalemia. √ Hypokalemia features include muscle weakness and cramping.
Key 28	**Paroxysmal Supraventricular Tachycardia = (Narrow-Complex SVT)** ◙ Usually in **young** patients ◙ Presents with **Palpitations**, **Light-headedness**, **Recurrent**, **Young**. ◙ *Management: imp √* ♠ **Initial line** → **Valsalva manoeuvre**, **Carotid massage**. ♠ **Not improved?** → Intravenous **adenosine** (**6mg** Rapid IV Bolus), still not improved? → give additional **12mg adenosine**, still not improved? → give another **12mg adenosine.**

N.B. **Adenosine** is contraindicated in **asthmatics**

→ **Verapamil (CCB)** is the preferred option in SVT in a patient with **Asthma**.

♠ **Still not improved?** → Electrical DC "**Cardioversion**"

• **Prevention of future episodes**

→ **ß-Blockers** or **Radio-frequency ablation.**

IMPORTANT NOTE:

If the patient is hemodynamically **unstable** (eg, hypotension, chest pain, shock, and or altered mental status)

Proceed immediately to → electrical synchronised cardioversion.

In summary:

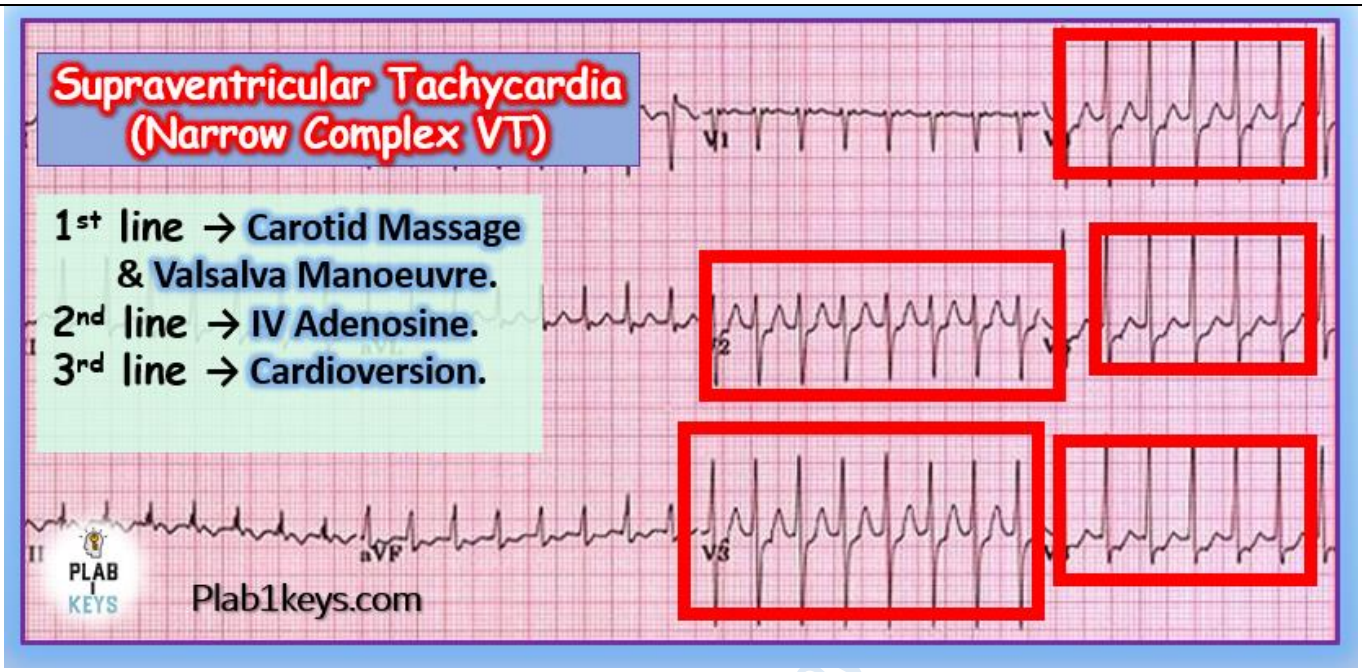
→ Carotid Massage and Valsalva Manoeuvre

→ IV Adenosine 6 mg

→ IV Adenosine 12 mg

→ IV Adenosine 12 mg

→ Cardioversion (Shock). [**Important Note**: Start with this step if chest pain, hypotension, and or altered mentation ie, hemodynamic unstable patient].



Polymorphic (Broad-Complex) Ventricular Tachycardia

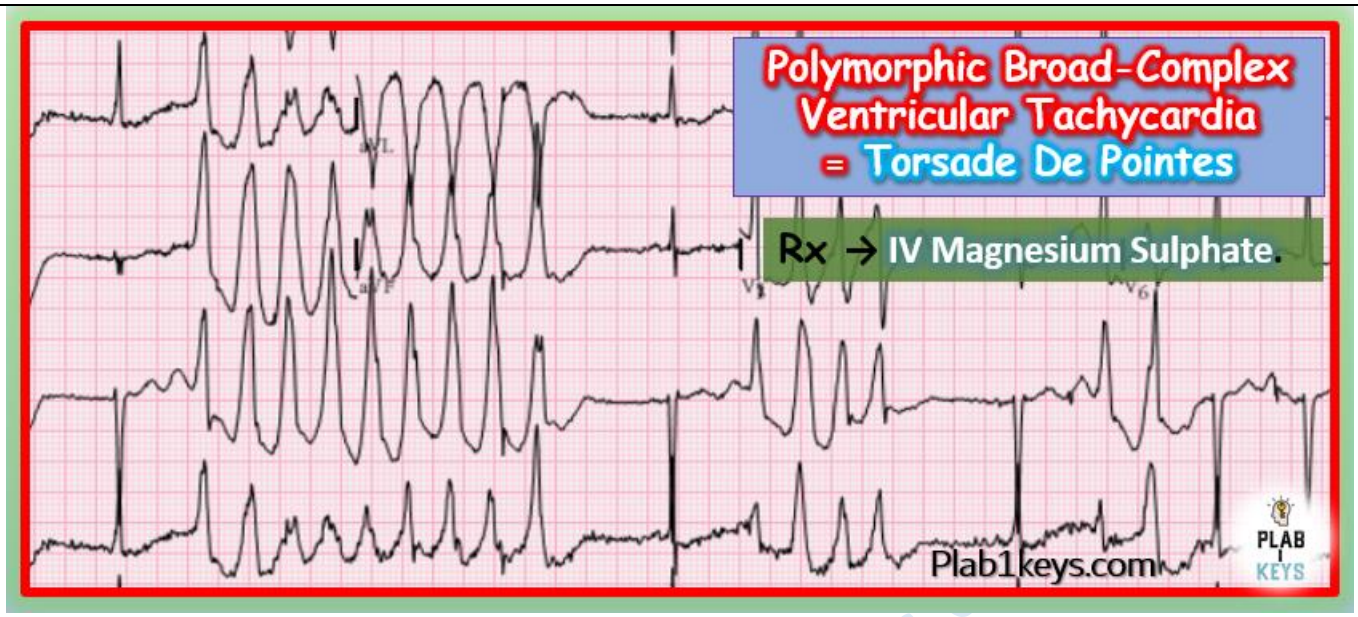
= Torsades De Pointes (TDP)

◙ Beat-to-beat variations with no uniform pattern of ventricular contractions.

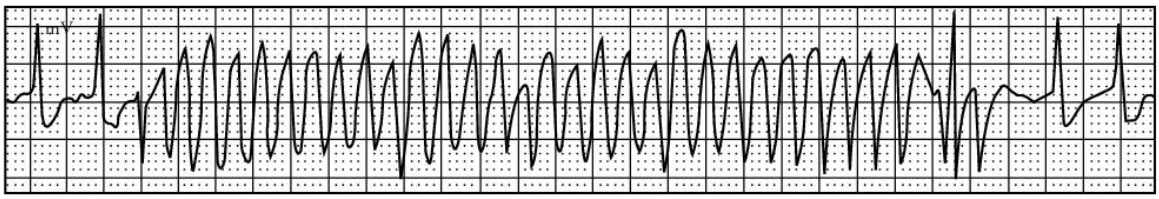
◙ **Broad QRS** (except in resting status), **Prolonged QT**, **Fainting episodes**, Patient might be a **young athlete**, **Recurrent**.

◙ **Treatment** → **IV Magnesium Sulphate**.

N.B. Verapamil should NOT be used in VT.



ECG Indicating Torsades de Pointes



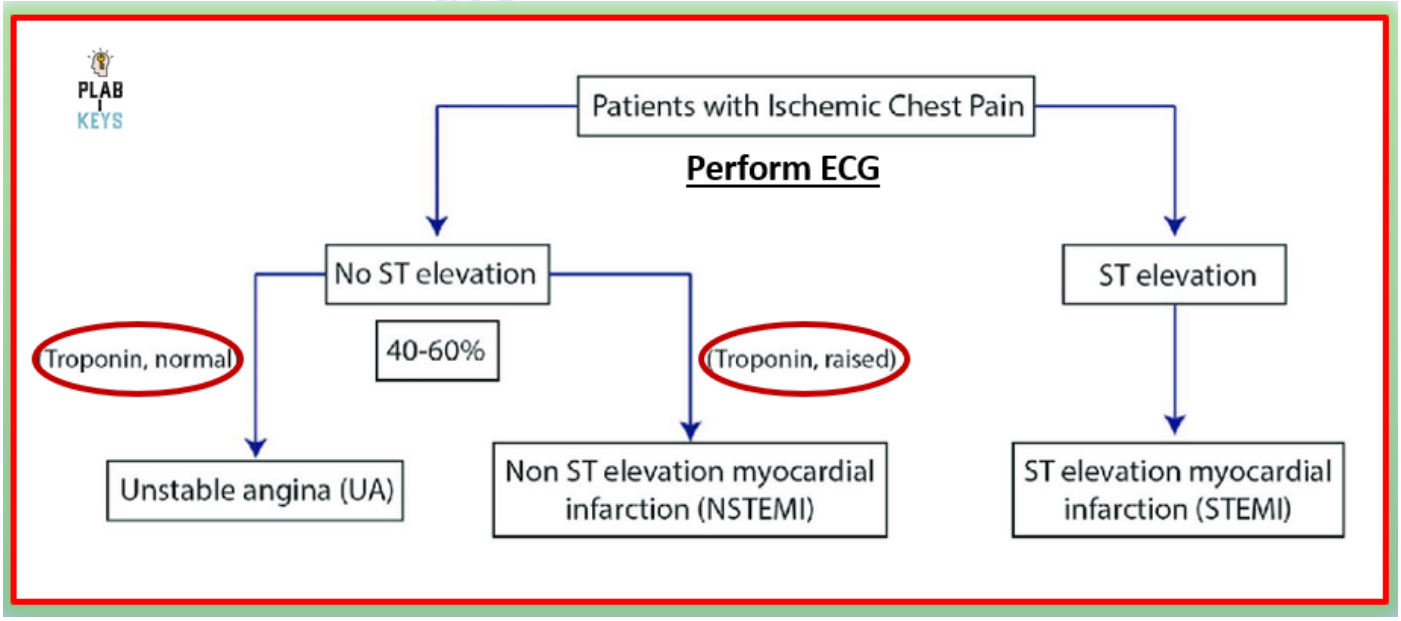
◙ **Quick Important Note to Remember:**

High doses of **Citalopram**, (a SSRI), can cause prolonged QT intervals, which can lead to **Torsade De Pointes**.

Key 29

For patients who present with STEMI, give MONA (Morphine, O2, Nitroglycerin, Aspirin) and send immediately for PCI (Percutaneous Coronary Intervention).

What if PCI is not given in the options?

	Pick → **Alteplase** "preferred" **or Streptokinase** (Tissue Plasminogen Activator) i.e. **Thrombolysis**.
Key 30	**MI (Acute chest pain radiating to jaw, shoulder…) BUT without ST elevation on ECG. What to Do Next?** → Request Cardiac Enzymes, especially (**Troponin**) If Troponin is high → **NON-STEMI Elevation MI** → **Give LMWH OR Fondaparinux** + **Aspirin 300 mg** 

Key 31	**6 weeks after MI, a patient returns with SOB when walking long distance and his ECG shows ST elevation in V1-V5 leads.** The likely cause → **Left Ventricular Aneurysm**. **(Persistent ST elevation post-MI → Think of: Left Ventricular Aneurysm)** **Left ventricular aneurysm** ◙ The ischaemic damage sustained during a MI episode may weaken the myocardium resulting in a thin muscular layer; thus, aneurysm formation. ◙ This usually **occurs 4-6 weeks post MI**. ◙ This is typically associated **with persistent ST elevation and left ventricular failure**. ◙ ***A thrombus*** *may form within the aneurysm increasing the risk of* ***stroke****. Patients are therefore* ***anticoagulated****.* ***ECG →*** ***Persistent ST Elevation*** *+ Left Ventricular Failure.* ***CXR →*** *Enlarged heart with a* ***bulge*** *at the left heart border.* ***Echo →*** *Paradoxical movement of the ventricular wall.*

Key 32

Hypertension Management

Hypertension classification

Stage	Criteria
Stage 1 hypertension	**Clinic BP** ≥ 140/90 mmHg and subsequent **ABPM** daytime average **or HBPM** average BP ≥ 135/85 mmHg
Stage 2 hypertension	**Clinic BP** ≥ 160/100 mmHg and subsequent **ABPM** daytime or **HBPM** average BP ≥ 150/95 mmHg
Stage 3 "Severe hypertension"	**Clinic** systolic BP ≥ 180 mmHg, **or clinic** diastolic BP ≥ 110 mmHg

Keys:

ABPM → Ambulatory Blood Pressure Monitoring.

HBPM → Home Blood Pressure Monitoring.

N.B. ***Clinic BP is usually higher than ABPM and HBPM because some people get stressed or feared while at a clinic → a slight increase in BP*****.**

Management of hypertension

Lifestyle advice should not be forgotten:

- Low salt diet.
- Caffeine intake should be reduced.
- Stop smoking.
- Drink less alcohol.
- Eat a balanced diet rich in fruits and vegetables.
- Exercise more.
- Lose weight.

♠ When to Treat Stage 1 Hypertension?

- Treat if the patient's age is < 80 years **AND** + any of the following:

Target organ damage, established cardiovascular disease, renal disease, diabetes (DM) or a 10-year cardiovascular risk equivalent to ≥ **10%.**

◙ **Note: If a patient is completely free and has a stage 1 Hypertension**

→ **Lifestyle and Diet Modification + review (Follow up).**

◙ **Note: In a patient with stage 2 hypertension at a clinic (Clinic BP ≥ 160/100)**

→ **Before commencing antihypertensive drugs, record either ABPM or HBPM.**

◙ **Note: For patients < 40 years and with stage 2 hypertension or higher**

→ **Consider a specialist referral to exclude secondary causes of the HTN**.

If ABPM or HBPM ≥ 150/95 mmHg (i.e., confirmed **stage 2 or higher hypertension**) ⟶ **Always treat**.

The Steps of The Management of Hypertension

Step 1

- Patients < 55-years-old ⟶ start with ACE inhibitor (**A**) or ARBs.
- Patients ≥ 55-years-old **or** of Afro-Caribbean origin "**of any age**"

→ start with Calcium channel blocker.

In other words:

- **White + < 55 YO** → start with **ACEi**/**ARBs** as a step 1 management of HTN.
- **White + > 55 YO** → start with **CCB** as a step 1 management of HTN.
- **Afro-Caribbean + any age** → start with **CCB** as a step 1 management of HTN.

Step 2 (still hypertensive after step 1)

• Both: **A**CE inhibitor + **C**alcium channel blocker (**A** + **C**)

Step 3 (still hypertensive after step 2)

• Add a Thiazide **D**iuretic (**D**)

So, 3 medications are taken → **A**CEi + **C**CB + Thiazide like **D**iuretic (**A**+**C**+**D**).

- Example of **ACEi** → Enalapril, Ramipril.
- Example of **ARBs** → Valsartan, Losartan, Candesartan.
- Example of **CCB** → Amlodipine, Felodipine.
- Examples of **thiazide diuretics** → chlorthalidone (12.5-25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily).
- ~~Bendroflumethiazide~~ is a thiazide like diuretic; however, it is no longer recommended by NICE as an antihypertensive.

Step 4 (For resistant hypertension) (For Reading)

• **consider further diuretic treatment.**

◙ If potassium < 4.5 mmol/l → add spironolactone (Potassium Sparing) 25mg OD.

◙ If potassium > 4.5 mmol/l → add a higher-dose thiazide-like diuretic.

• If further diuretic therapy is not tolerated or is contraindicated or ineffective, consider an alpha- or beta-blocker.

■ **Patients who fail to respond to step 4 measures should be referred to a specialist**.

■ NICE recommend: If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of **four drugs** → **seek expert advice**.

Blood pressure targets

◙ ***For Diabetic patients with Hypertension:***

If end-organ damage (e.g. renal disease, retinopathy) < 130/80 mmHg otherwise < 140/80 mmHg.

◙ ***For Hypertensive patients without DM:***

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Hypertension + Diabetes (V. Imp)

◙ Always treat hypertension in a **DIABETIC** patient with **ACE inhibitor** regardless of the age as it is reno-protective "Unless if the eGFR is <30".

◙ However, if this diabetic patient is **Afro-Caribbean**,

→ start with both **ACE inhibitor + Calcium Channel Blocker** as a first step.

■ **Before commencing ACE inhibitor for any patient → check eGFR.**

If eGFR (Glomerular Filtration Rate) is low; <30 as in advanced Chronic Kidney Disease ⟶ ACEi and ARBS should be avoided in this case.

Why ACE inhibitor is used for Diabetic Hypertensive patients?

- It is **reno-protective** (unless eGFR is low; <30; in advanced CKD).
- It has **protection** against diabetic **retinopathy**.
- It has **+ve effect** on **glucose metabolism**.

Key 33

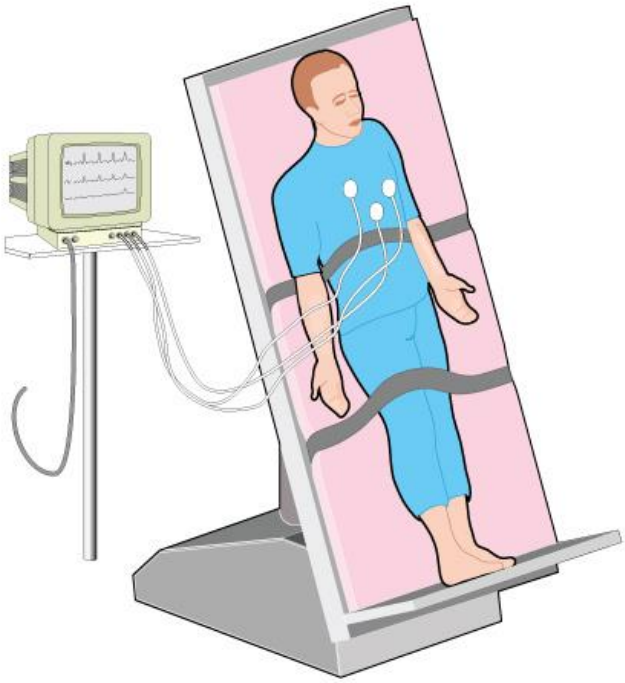
Postural Hypotension (Orthostatic Hypotension)

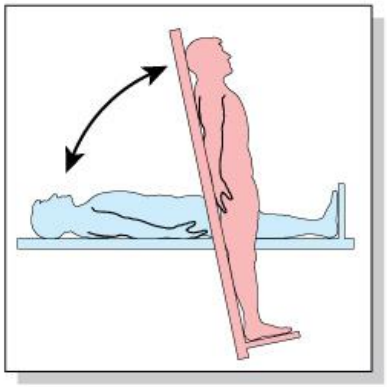
- A **drop** in **systolic** blood pressure of at least **20** mm Hg within **three** minutes of standing.
- or a **drop** in **diastolic** blood pressure of at least **10** mm Hg within **three** minutes of standing.

- BP is measured on lying position, then on standing position.

- Dx: **Monitor BP.**
- Other useful and important test → **Tilt table test**.

A tilt table test is used to evaluate the cause of unexplained fainting. A health care provider might recommend a tilt table test to evaluate repeated, unexplained episodes of lightheadedness, dizziness or fainting. The test can help determine if the cause is related to heart rate or blood pressure.





◙ Postural hypotension is common in **elderly people** especially those who take **multiple drugs** (**Polypharmacy**) and those with **hypertension**.

◙ Anti-hypertensive medications can cause postural hypotension as well (eg, **ACE inhibitors** as **Ramipril**). → This could lead to **recurrent falls**.

◙ The **Baroreflex mechanisms** that control HR (Heart Rate) and VR (Vascular resistance) decline with age, particularly in patients with hypertension.

Q] An elderly man complains of difficult mobilisation. He often feels dizzy upon trying to stand ± He has a Hx of Recurrent Falls. Management?

→ **Blood pressure monitoring** & **Assess and review the patient's Medications**.

Q] An elderly man takes several medications for hypertension and heart failure. He often feels dizzy upon trying to stand ± He has a Hx of Recurrent Falls?

The likely cause of his postural hypotension → **polypharmacy**.

Management → **Blood pressure monitoring**.

Q] An elderly man feels dizzy when he stands and loses balance and falls. He is on a number of medications: ramipril for hypertension, zopiclone insomnia, cetirizine for allergy of hay fever, metformin for diabetes, and lithium for bipolar disorder. He has postural hypotension (his diastolic BP while standing is > 10 mmHg lower than his diastolic BP while sitting). Which medication is the most likely reason for his recurrent falls?

→ **ACE inhibitors (Ramipril)**. **They can cause postural hypotension → thus falls.**

Anti-hypertensive medications as ACE inhibitors and Calcium channel blockers can cause postural hypotension → dizziness on standing → Falls.

	Note: if there was no postural hypotension, we would suspect **zopiclone** (Although rare, it can lead to falls by balance impairments).
Key 34	Again, **any patient of any age and any ethnic group presents with <u>Hypertension</u> and he is a <u>Diabetic</u> patient** Start with → **ACE inhibitor** (e.g. Enalapril). (**Note**, if the eGFR is 30 or below, ACEi and ARBS should be avoided).
Key 35	**Absent "P" wave on ECG** + **Irregularly Irregular Rhythm** + **Palpitation** **Diagnosis?** → **Atrial Fibrillation**. **Management?** √ First line → beta-blockers. √ If asthmatic → Avoid beta-blockers and give calcium channel blockers. √ If the patient has associated HF → give digoxin. **+** → Calculate **CHA2DS2-VASc Score** **(Key number 18) and accordingly:** **→ Give (Warfarin) or (DOAC) or nothing according to the Cha2ds2vasc score.**

	◙ **Examples of DOAC "D**irect-Acting **O**ral **A**nti**C**oagulants" -**important** √- **Apixaban**, **Rivaroxaban**, **Edoxaban**, **Dabigatran**". √ **If haemodynamically unstable** (eg, SBP ≤ 90) → **Cardioversion** (Shock). √ If the patient with AF is **unstable** (eg, hypotension) and the AF has **just started** and no cardioversion in the options, Pick → **IV amiodarone**. **Imp √**
Key 36	**Fever + New Murmur** → **Infective Endocarditis** "until proven otherwise". Be careful, the reason of IE is → **Infection** "infective" endocarditis. - **Staph. Aureus** is the commonest cause of IE **in general**. - **Staph**. **Epidermidis** is the commonest cause after **prosthetic valve surgery.** - **Strept**. **Viridans** (especially sterpt. Mitis and strept. Sanguinis) are the commonest cause in people with poor dental hygiene or following a **dental procedure**.
Key 37	**Ventricular Ectopics** **= Three-beat patterns = Ventricular Trigeminy.**

	◙ A sense of a **missed**/**skipped beat**, **unsustained palpitation** ± Dyspnea and Dizziness due to immature discharge of a ventricular ectopic focus which produces → an early and **broad QRS** complex. ◙ **Causes →** Ischemic heart disease (MI), Cardiomyopathy, Stress, Alcohol, Caffeine, Cocaine, Medications **OR** Naturally. ◙ Over half the population have silent, or asymptomatic ventricular ectopics which are discovered incidentally on a routine ECG. ◙ If there is No underlying Heart disease (e.g. IHD, Cardiomyopathy) → **Benign**; no clinical significance. ◙ If these ventricular ectopics are due to IHD or Cardiomyopathy → May precipitate to more life-threatening arrhythmias like **Ventricular Fibrillation**.
Key 38	**The Typical Presentation of Acute MI (75% of cases)** ◙ Central Chest Pain or Epigastric or Substernal pain that is severe, sudden, crushing, pressuring, squeezing or burning and radiates to arms, shoulders, neck or jaw. ◙ ± Sweating (Diaphoresis), Nausea, Vomiting, Fatigue and/or Palpitations.

	◙ SOB "Shortness of breath". ♠ **Keep in mind that some patients may present with additional Atypical feature such as Abdominal Pain, Jaw pain or Altered mental status.**
Key 39	**Long term medications post-Myocardial Infarction = 5 Drugs** *Aspirin, Clopidogrel, ß- Blockers, ACEi, Statins* **AABCS** → **A**spirin, **A**CEi, **B**B, **C**lopidogrel, **S**tatin (e.g. Atorvastatin)
Key 40	**A patient with chronic heart failure developed gout. A medication for his gout is prescribed. A few days later, the patient came back to the hospital complaining of worsening of his Heart Failure symptoms (SOB, Orthopnea).** - The likely cause of this patient's **gout** → **Thiazide like diuretics (e.g. bendroflumethiazide)** or **Loop Diuretics** (Both can cause hyperuricemia (Gout) and both can be used to treat volume overload caused by Heart Failure) - The likely cause of this patient's **worsening of SOB and Orthopnea** → **NSAIDs** (e.g. **Ibuprofen**) that was prescribed to treat his gout.

Important Notes

◙ Never give **NSAIDs** (e.g. Ibuprofen) nor **selective COX-2 inhibitors** (e.g. Celecoxib) to the following patients: **CKD**, **CHD**, **IHD**

(Chronic Kidney Disease, Chronic Heart Failure, Ischemic Heart Disease).

◙ These drugs can worsen the HF (worsening the SOB and Orthopnea) and also the renal function.

◙ Remember that NSAIDs **inhibit** the synthesis of **prostaglandins**

→ thus, decrease the eGFR, retain more salt and water (risk factor for HF).

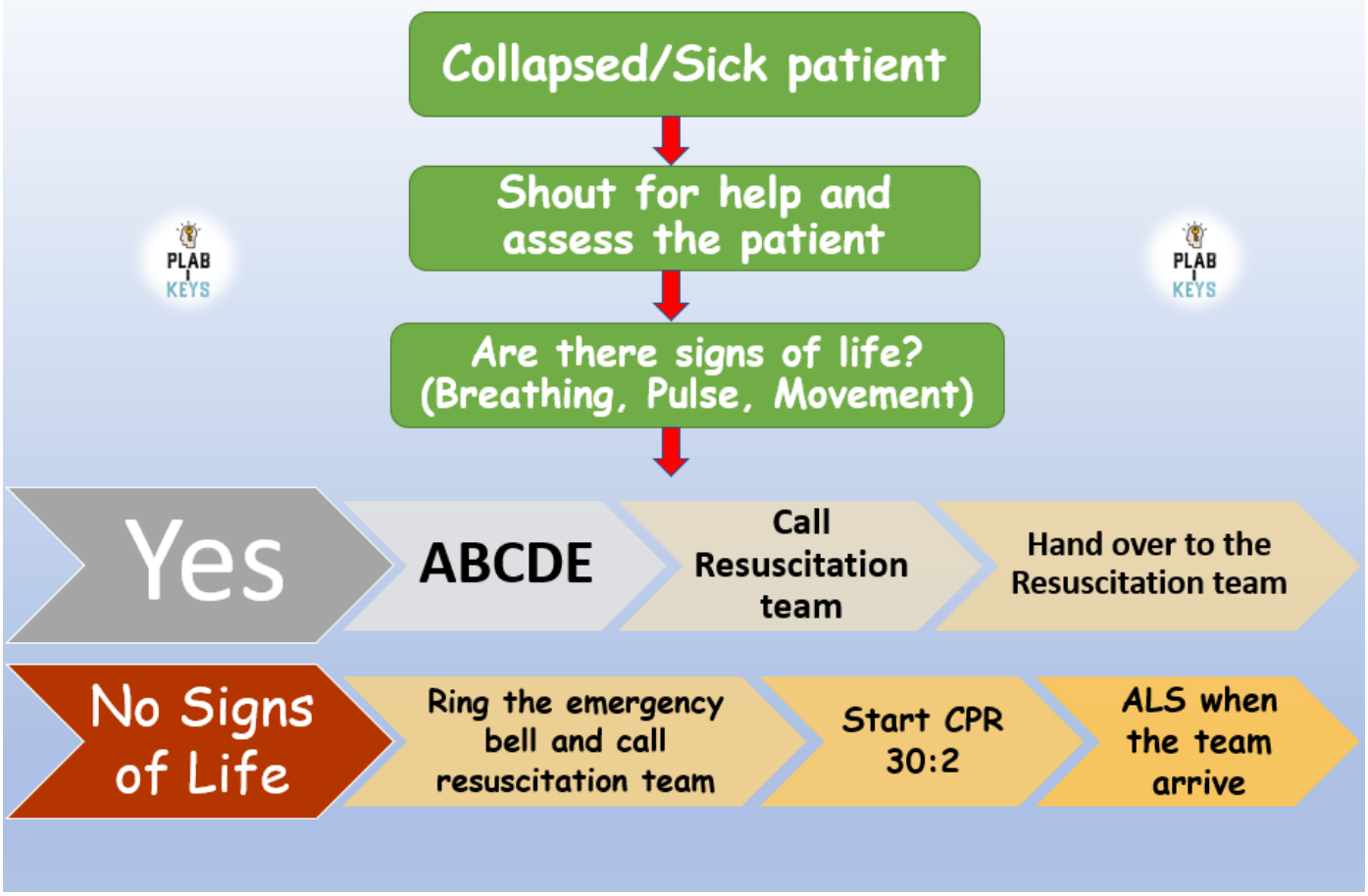
◙ N.B. **Thiazide like diuretics** and **Loop diuretics** decrease the clearance of Uric Acid → leading to **Gout** **(Hyperuricemia)**

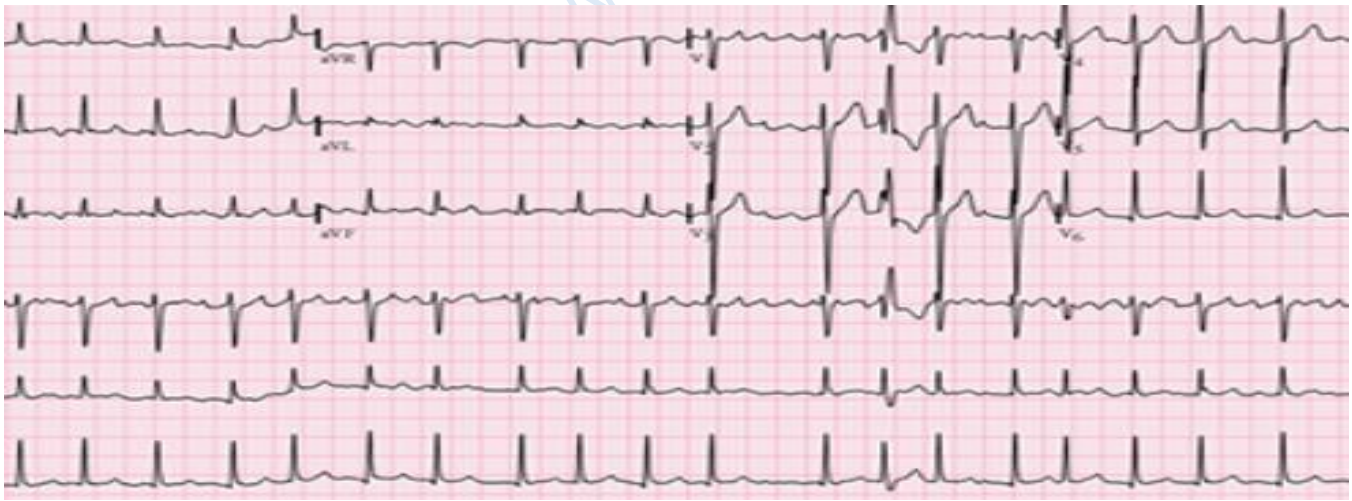
◙ N.B. NSAIDs such as **Ibuprofen** are used for the treatment of Gout. If given to a patient with chronic heart failure, they would worsen the HF symptoms (Orthopnea and Dyspnea).

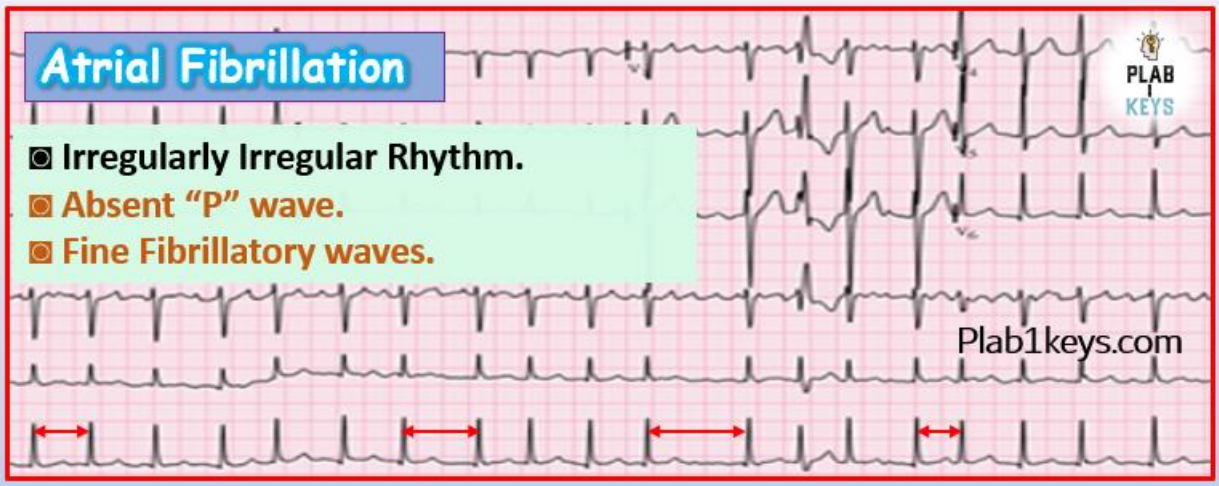
Key 41

In-Hospital Cardiac Arrest algorithm

If No Signs of Life (i.e. No breathing, No detectable Pulse):

	1) **Ring the emergency bell and call resuscitation team** (Code Blue) first. Then → 2) **Start CPR 30:2. Then →** 3) **Get defibrillator. Then →** 4) **ALS when the resuscitation team arrives**. 
Key 42	**<u>In STEMI patient, what if PCI is not given in the options?</u>** Pick → **Alteplase** "preferred" **or** **Streptokinase** (Tissue Plasminogen Activator) = i.e. **Thrombolysis**.

Key 43	**Diabetic patients may develop "Silent MI" i.e. painless MI. Thus, they may die suddenly and silently without feeling any chest pain (They won't feel chest pain → They won't seek medical help).** **This is because they may not feel chest pain due to autonomic neuropathy.**
Key 44	**A scenario to test your knowledge on a previous topic** **An elderly male presents with Palpitations and Shortness of breath on exertion. The ECG is as follows.** **What is the likely diagnosis and management?**  **Answer:**



Palpitation, Tachycardia, Dyspnea (**SOB**), Fibrillatory waves on the ECG, Irregularly Irregular Rhythm ± Absent "P" wave.

→ **Atrial Fibrillation** → Give **Beta-Blocker.**

If Asthmatic → Give **Calcium Channel Blocker.**

Key 45

Remember:

In **Supraventricular tachycardia** (Narrow QRS Complex, absent p waves)

→ We **firstly** perform **Carotid Massage** and **Valsalva Manoeuvre**.

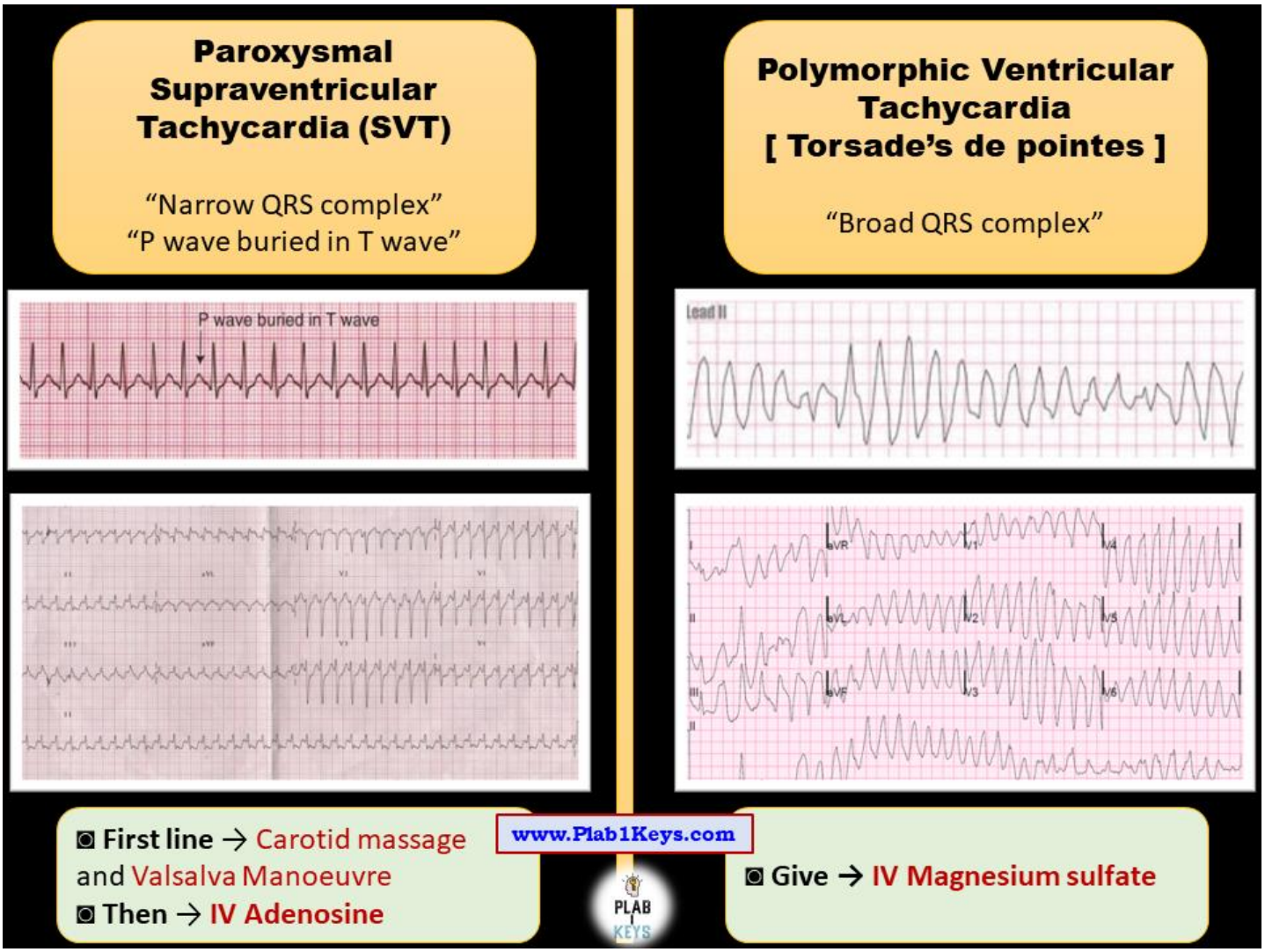
If this fails → We give **IV Adenosine**.

IMPORTANT NOTE:

If the patient is hemodynamically **unstable** (eg, hypotension, chest pain, shock, altered mental status)

Proceed immediately to → electrical synchronised cardioversion.

	◙ In **polymorphic ventricular tachycardia** (i.e. torsade's de pointes) → **IV magnesium sulfate**.
Key 46	**Beck's Triad in Cardiac Tamponade** **Hypotension**, **Muffled Heart Sounds**,



High JVP (**Distended neck veins**).

- **Trauma** (e.g. stab in the chest) is the most important cause for cardiac tamponade.

- Dx: **Echocardiography** is diagnostic.

- Tx: **Urgent pericardiocentesis**.

Key 47

Remember that:

◙ In **Atrial Myxoma** → Mitral valve obstruction → **Mitral Stenosis**

→ **Early or Mid-diastolic murmur**, **Dyspnea**, **Syncope**

◙ In **Atrial Myxoma** → Breakdown of small emboli from the mass that can travel down the blood and cause ischemia in multiple sites, leading to

→ (e.g. **Pulmonary Embolism**, **Stroke**, **Clubbing**, **Blue fingers**)

	Therefore, in a patient with Hx of syncope, SOB, Pulmonary Embolism and early-mid diastolic murmur → Think of **Atrial Myxoma**.
Key 48	**Points on Alcohol** UK guidelines recommend that a person should drink - **No more than 14 units a week,** - **No more than 3 units a day,** - **with at least 2 alcohol-free days a week.** **Example:** **If someone drinks 7 units of alcohol a week and smoke 20 cigarette a day, we should refer him to** **→ Smoking Cessation Clinic.** **This is because his alcohol intake is insignificant as per NICE whereas his smoking is significant.**
Key 49	**A scenario to test your knowledge on a previous topic** **4 days after MI, an elderly patient presents with Fatigue and Dyspnea. On Auscultation → Pansystolic murmur at the apex and radiates to the axilla was heard.**

→ The likely Dx → **Mitral Regurgitation**.

→ The likely Cause → **Rupture of Papillary Muscles**.

Acute mitral regurgitation (MR): "important √" pansystolic murmur

◙ Occurs 2-15 days after the MI (Mostly inferior MI).

◙ Due to ⟶ Ischemia or rupture of the papillary muscles of the mitral valve.

◙ An early-to-mid systolic or **Pansystolic murmur** is typically heard.

◙ May present with Hypotension, Tachycardia and Pulmonary edema.

◙ Dx ⟶ **Echocardiogram**.

◙ Treatment ⟶ vasodilator therapy but often requires emergency surgical repair

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Key 50

A scenario to test your knowledge on a previous topic

2 days after MI, an elderly patient presents with fever and chest pain. ECG shows ST elevation with upward concavity.

→ **Acute Pericarditis**.

	Post-MI Pericarditis "important √" ◙ Occurs within **2-3 Days** after MI. ◙ **Features → Pleuritic chest pain that is worse on lying flat and during inspiration ± Fever ± pericardial rub** ◙ Pericardial effusion may develop leading to enlarged globular heart on chest X-ray and is confirmed by echocardiogram. ◙ **ECG → Widespread Saddle Shaped ST Elevation** with **upward concavity** + PR Depression. ◙ **Management of Pericarditis (imp):** √ A full-dose **NSAID** should be used (eg, aspirin, 2-4 g/d; **ibuprofen** 1200-1800 mg/d; indomethacin 75-150 mg/d); treatment should last at least 7-14 days. √ **Colchicine** (as an adjunct to NSAIDs to ↓ inflammation and recurrence).
Key 51	**For Acute Myocardial Infarction patients, the analgesic that can be used while in the ambulance is still → IV Morphine.** ◙ Remember the initial management for acute MI → (**MONA**): **M**orphine, **O**xygen, **N**itrates, **A**spirin. --- **MI Analgesia while in an ambulance (Pre-Hospital)** 1) Glyceryl Trinitrate (**GTN**) sublingual or spray.

2) ± **Opioids** (**INTRAVASCULAR**): 2.5-5 mg Diamorphine or 5-10 mg Morphine.

- **N.B**. Around **1/3** of the patients with MI have **nitrate-resistant** chest pain; therefore, morphine is given additionally to relieve chest pain.

- **Why IV and not IM**?

√ IM absorption is unreliable **+**

√ If the patient receives thrombolysis later on, the site of IM injection might bleed.

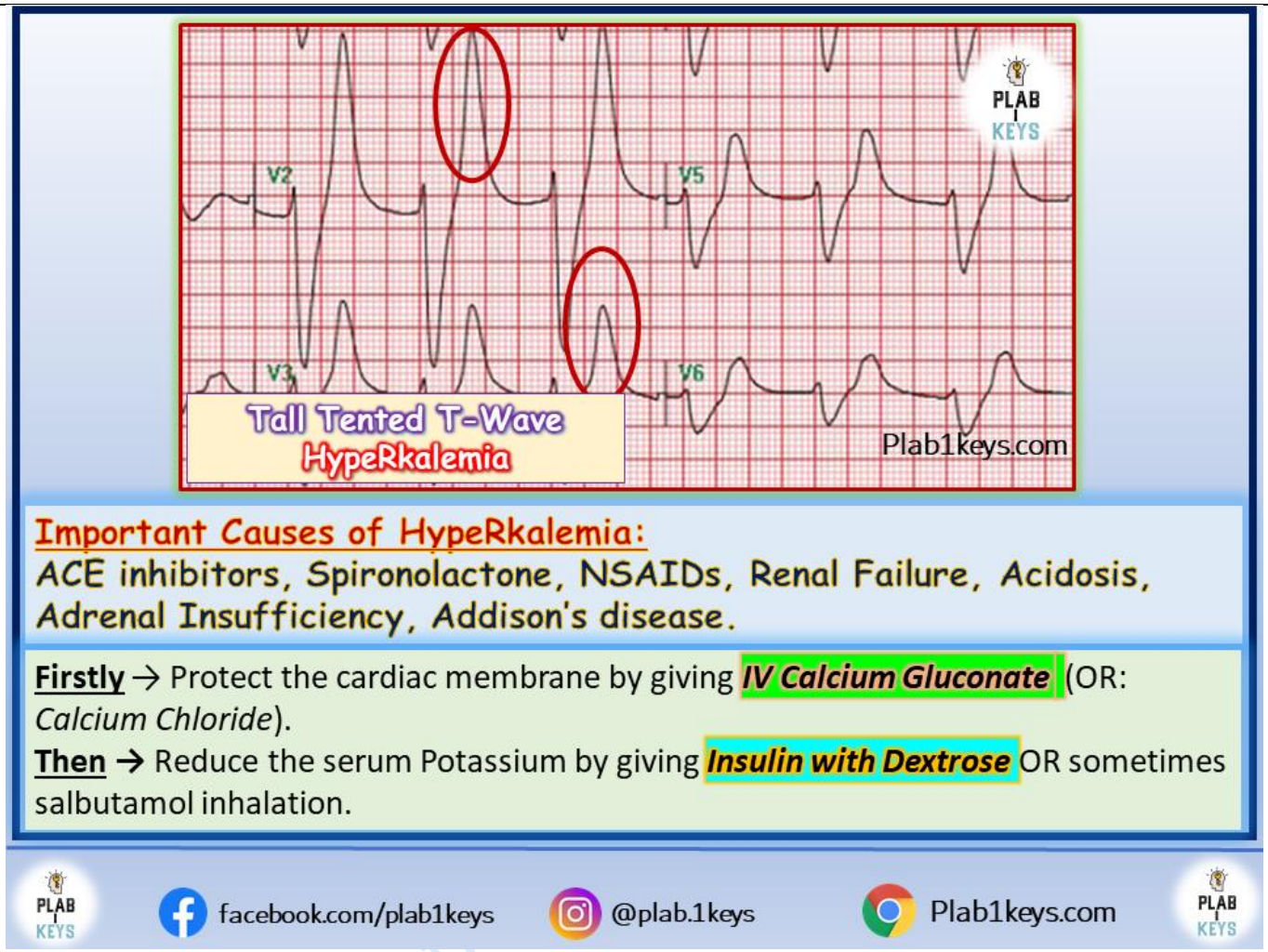
Key 52

Ventricular Fibrillation	**Old adult, Sudden collapse, Not breathing, Unconscious →** Shock ''defibrillation''

Broad Complex Tachycardia

◙ Tachyarrhythmia is one of the complications of MI.

	♠ An ECG showing broad complex tachycardia in a (**still**) **conscious** patient ± atrial activity → **Ventricular tachycardia** → **IV Amiodarone**. ♠ If the patient is **hemodynamically unstable**; i.e. unconscious, collapsed, not breathing → **Ventricular Fibrillation** → 1) Check the patient's pulse, if **no pulse**, commence the **arrest protocol immediately**. 2) Administer **O2**. 3) If the patient is hemodynamically unstable: **Shock** followed by **IV Amiodarone** followed by **further Shocks** if needed. ◙ **N.B. HypOkalemia** is the **most important cause of ventricular tachycardia (VT) clinically.**
Key 53	Treatment of Cardiac Tamponade → **Pericardiocentesis**.
Key 54	◙ **Tall Tented T**-wave → **HypeRkalemia**. ◙ **U**-wave → **HypOkalemia**.



Key 55

A scenario to test your knowledge on a previous topic

45 Y/O African patient has BP 160/90 on three separate occasions.

What is the initial line of treatment?

→ **Calcium Channel Blocker (CCB)**

(**First Step Management of hypertension in African-Caribbean patients is CCB regardless of the age**).

- **White + < 55 YO** → start with **ACEi/ARBs** as a step 1 management of HTN.
- **White + > 55 YO** → start with **CCB** as a step 1 management of HTN.
- **Afro-Caribbean + any age** → start with **CCB** as a step 1 management of HTN.

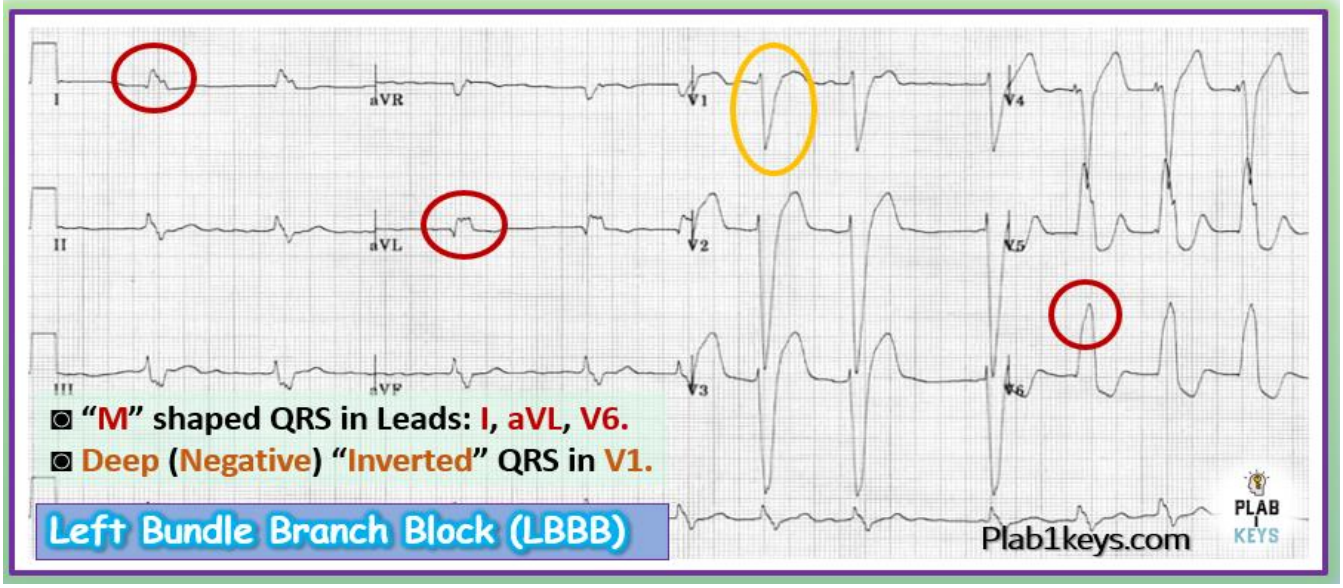
- **Any ethnicity + Any age + Diabetes**

→ start with **ACEi/ARBs** as a step 1 management of HTN

Key 56

Remember that

LBBB is associated with acute MI.



Key 57

Heart Murmurs

Defect	Type of Murmur	Where is it Best heard?	Symptoms
Aortic Stenosis	Ejection Systolic	Right 2nd ICS just lateral to sternum, radiates to Carotid artery	Dyspnea on activity, Anginal chest pain, syncope
Aortic Regurgitation	Early Diastolic	left lower sternal border around the 3rd to 4th intercostal spaces	Symptoms of Heart Failure
Pulmonary Stenosis	Ejection Systolic	Left 2nd ICS just lateral to sternum, radiates to left shoulder of infraclavicular area	Systemic Cyanosis
Pulmonary Regurgitation	Early-Diastolic	Left 2nd ICS just lateral to sternum	Symptoms of Right-Sided Heart Failure
Mitral Stenosis	Mid-Late Diastolic, with opening click	Apex (left 5th ICS midclavicular line)	Symptoms of Heart Failure

Mitral Regurgitation	Pan-Systolic	Apex (left 5th ICS MCL), radiates to Axilla	Symptoms of congestive Heart Failure; edema, Ascites
Tricuspid Stenosis	Diastolic Rumble	4th-5th ICS over the left sternal border.	Fluttering Discomfort in the neck
Tricuspid Regurgitation	Pan-Systolic © Copyright www.plab1keys.com	4th-5th ICS over the left sternal border.	Symptoms of Right-Sided Heart Failure

Example:

A patient with a history of MI presents with Orthopnea (Cannot lie down flat), Bibasilar crepitations, Pan-systolic murmur.

→ **Mitral Regurgitation.**

→ Request **Echocardiogram.**

Key 58

A scenario to test your knowledge on a previous topic

A Young adult presents with frequent fainting attacks since childhood and prolonged QT. There are sinus rhythm and normal P-R interval. No FHx of arrhythmias or sudden death.

The likely Dx→ **Polymorphic Ventricular Tachycardia (Torsades de pointes)**

Polymorphic (Broad-Complex) Ventricular Tachycardia

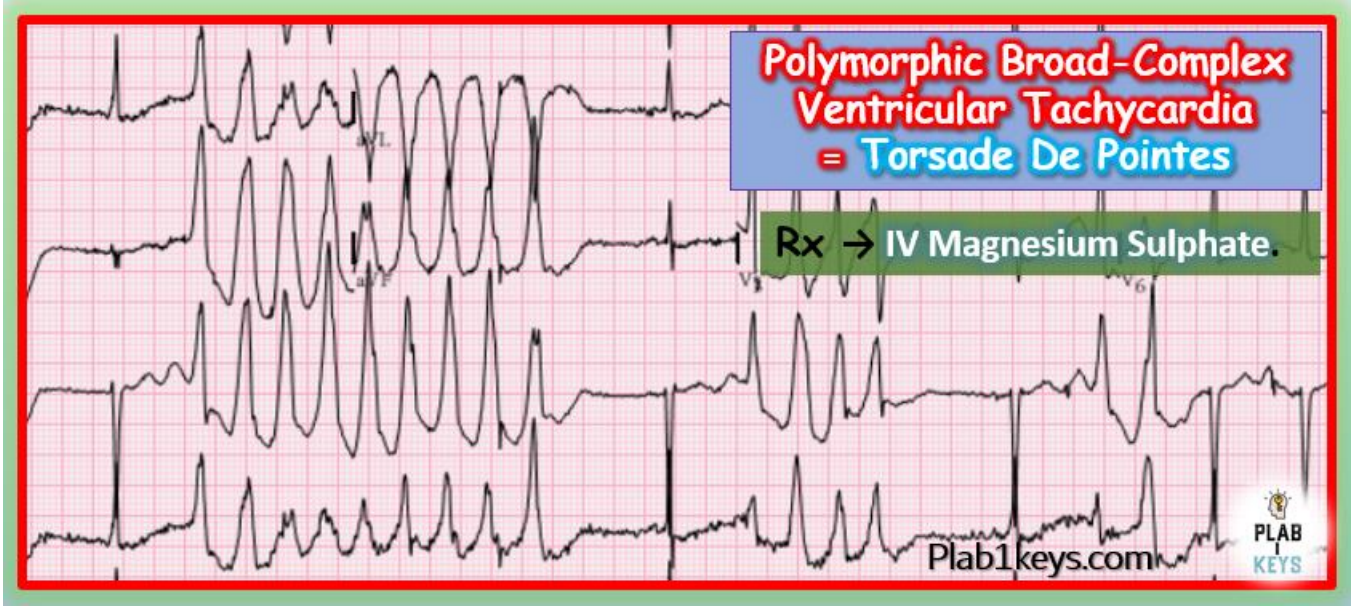
= Torsades De Pointes (TDP)

◙ Beat-to-beat variations with no uniform pattern of ventricular contractions.

◙ Broad QRS (except in resting status), Prolonged QT, Fainting episodes, Patient might be a young athlete. Recurrent.

◙ **Treatment** → **IV Magnesium Sulphate**.

N.B. Verapamil should NOT be used in VT.



Key 59

An elderly patient with a Hx of stroke presents with exertional dyspnea.

ECG shows Atrial Fibrillation. Chest X-ray shows Straight left heart border.

→ **Mitral Stenosis**.

- The most common cause of mitral stenosis → **rheumatic fever**, **rheumatic fever** and **rheumatic fever**.

Pathogenesis of Mitral Stenosis:

Mitral stenosis impedes left ventricular filling → increased left atrial pressure (Which will lead to **left atrial hypertrophy**; therefore, CXR shows **Straight** left side heart border) → Blood returns Back to lungs → Pulmonary Congestion → Right Ventricular Failure (Hepatomegaly, Ascites, Oedema)

Features

- **Mid-late diastolic murmur** (best heard on **expiration**) ''low pitched''
- **Loud S1**, **opening snap**
- Low volume pulse
- Malar flush
- **Atrial fibrillation**

Note:

◙ Left heart murmurs (**Mitral** and **Aortic**) are best heard during expiration

◙ Right heart murmurs (**Tricuspid** and **Pulmonary**) best heart in inspiration

	Careful! Left and right refer to the site of the valve, not the area of auscultation **Features of severe MS** • The length of murmur increases • The opening snap becomes closer to S2 **Chest x-ray in MS** • **Left atrial enlargement** (often) → **Straightening the left border of the heart**. **ECG** (may show): - Signs of Right ventricular hypertrophy - **P mitrale** (bifid P wave) - **AF** **Echocardiography** → (Thickening of Mitral valve leaflets)
Key 60	First line treatment in AF (if no asthma) → **ß-Blockers (e.g. Metoprolol)**

Key 61

◙ ↓ Ejection Fraction (+) ↓ Septal Wall Thickness →

Dilated Cardiomyopathy

◙ ↑ Ejection Fraction (+) ↑ Septal Wall Thickness →

Hypertrophic Cardiomyopathy.

Dilated Cardiomyopathy (DCM) Basics (For READING ONLY)

- Dilated heart leads to systolic (± diastolic) dysfunction
- All 4 chambers are affected but the Left Ventricle is more affected than the Right Ventricle.
- Features include arrhythmias, emboli, mitral regurgitation
- Absence of congenital, valvular or ischaemic heart disease

◙ Causes often considered separate entities

- alcohol: may improve with thiamine
- postpartum
- hypertension

◙ Other causes

	• **inherited** • previous MI • infections e.g. Coxsackie B, HIV, diphtheria, parasitic • endocrine e.g. Hyperthyroidism • infiltrative e.g. Haemochromatosis, sarcoidosis • neuromuscular e.g. Duchenne muscular dystrophy • nutritional e.g. Kwashiorkor, pellagra, thiamine/selenium deficiency • drugs e.g. Doxorubicin **◙ Inherited dilated cardiomyopathy** • around a third of patients with DCM are thought to have a genetic predisposition • a large number of heterogeneous defects have been identified • the majority of defects are inherited in an **autosomal dominant** fashion although other patterns of inheritance are seen
Key 62	**A Summary on Arrhythmias Management** **◙ In Supraventricular tachycardia (Narrow-Complex) (SVT):**

♠ If the patient is **haemodynamically stable** → start with **Valsalva manoeuvre** and **Carotid massage** to stimulate the vagal tone (Parasympathetic which decreases the heart rate).

- If still ill? → Give **Adenosine 6 mg** IV bolus.
- If no response? → give another **Adenosine double dose (12 mg).**
- If still no response? → give another **Adenosine double dose (12 mg)**.
- Unsuccessful yet? → **Cardioversion**.

♠ If the patient is **haemodynamically Unstable** → start with **Cardioversion**.

◙ In Polymorphic Ventricular Tachycardia (Broad-Complex) = (Torsade De Pointe)

→ Give **IV MgSO4** (Magnesium Sulphate)

◙ In AF:

- Start with **ß-Blockers** (e.g. Metoprolol).
- If Asthmatic patient → **Calcium Channel Blockers**.
- If Associated Heart Failure → **Digoxin**.

◙ In Ventricular Tachycardia →

	If stable patient → **IV Amiodarone**. If unstable → **Synchronized cardioversion** (Shock). --- **Haemodynamically Unstable** → e.g. √ **Hypotension (SBP < 90)**, √ **Loss of Consciousness**, **Others**: √ HR > 150. √ Severe **dizziness** or syncope. √ **Ongoing chest pain**. √ increasing SOB. → **Synchronized DC Cardioversion**. [Any arrhythmia in an unstable patient → DC Cardioversion]
Key 63	**An elderly patient suddenly fell unconscious, he recovered completely within a few minutes, he remembers the event very well, he did not trip, he felt hot and flushed after the episodes but he did not feel dizzy or sweaty before the fall.**

The best Investigation → **12-lead ECG**

Analysis and Causes of Falls.

Causes are usually:

1) **Cardiac cause** (e.g. **Arrhythmia**).
2) **Postural (Orthostatic) Hypotension.**
3) **Hypoglycemia**.
4) **Seizure**.

◙ If the cause was **hypoglycemia**, he would have felt sweaty and dizzy before the episode. Plus, he would not have recovered until Glucose is administered.

◙ If it was a **seizure**, the stem has to include an eyewitness to describe the episode. Also, in seizure, there are usually post-ictal features such as confusion and drowsiness. However, the patient in the above stem did not feel any of these, he rather recovered completely.

◙ If it was **Postural hypotension** (Orthostatic hypotension), the fall usually follow a standing from a sitting position. In addition, the patient would have felt dizziness before the fall, Hx of difficult mobilisation, which are not mentioned here.

◙ Therefore, the likely cause here is **arrhythmia (Cardiac cause), likely (Stokes Adam attack).** This is also supported by the fact that the patient felt hot and flushed after recovery, which means that the blood has been, rapidly, pumped back to the already dilated vessels. (Dilated due to hypoxia caused by the irregular rhythm) → **12-lead ECG monitoring is required.**

Stokes Adam attack [For Reading]:

Sudden collapse into unconsciousness due to a disorder of heart rhythm in which there is a slow or absent pulse resulting in syncope (fainting) with or without convulsions. In this condition, the normal heartbeat passing from the upper chambers of the heart to the lower chambers is interrupted. This results in a condition called a "heart block." When a heart block occurs, the heart rate usually slows considerably. This can cause inadequate blood flow to the brain and result in fainting.

An elderly patient fell and collapsed "syncope". He was transferred to the A&E and now he is fully conscious. ECG shows irregular rhythm. What is the next best **investigation**?

→ **Echocardiogram**.

◙ A **Holter ECG** (**24-hour ECG**) will not be beneficial as the ECG already shows Irregular Rhythm; hence, there is no point of using it again.

	◙ **Echo** should be done to identify the **underlying cause** of this irregular rhythm so the treatment can be decided accordingly.
Key 64	**Murmurs in Paediatrics** - **Preterm baby with continuous or machinery murmur** → **PDA** (Patent Ductus Arteriosus). - **Cyanotic baby with ejection systolic murmur** → **TOF** (Tetralogy of Fallot). Ejection systolic murmur here is due to pulmonary stenosis which is one of the four major features of TOF. - **Progressive (Severe) Cyanosis + Poor feeding + Holosystolic "pansystolic" murmur along the left sternal border** → **Tricuspid Atresia**. - **Acyanotic + Pan-systolic murmur** → **VSD** (Others: Poor feeding and poorly gaining weight) **Congenital Heart Disease**

Cyanotic Congenital Heart Disease (R → L)

[5T's with 1-5 mnemonic]

Truncus arteriosus	Vessels join to make **1**
Transposition of great vessels	**2** major vessels switched
Tricuspid atresia	**3** (tricuspid)
Tetralogy of Fallot	**4** defects
Total anomalous pulmonary vascular return	**5** letters (TAPVR)

Acyanotic Congenital Heart Disease (L → R)

Atrial septal defect (ASD)

Ventricular septal defect (VSD)

Patent ductus arteriosus (PDA)

Coarctation of aorta (CoA)

It is important to remember that:

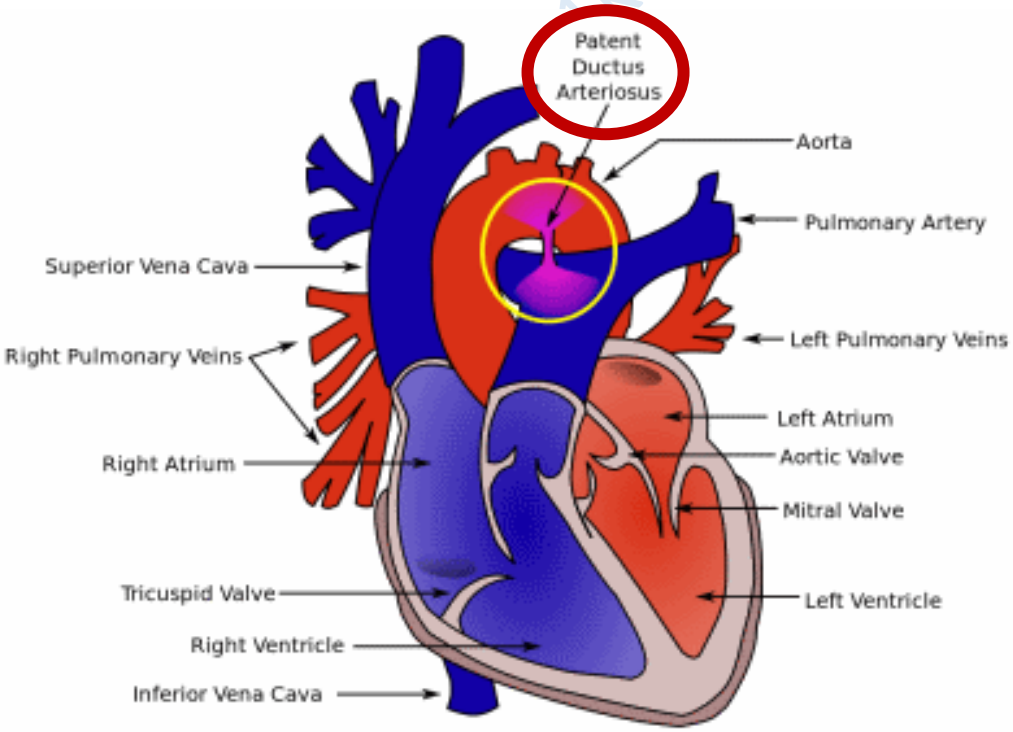
√ PDA, ASD, VSD → **Acyanotic**.

√ TOF, Tricuspid Atresia → **Cyanotic**.

√ Pansystolic murmur = MR or TA or VSD.

Patent Ductus Arteriosus (PDA)

- A form of congenital heart defect
- generally classed as '**acyanotic**'. **(√)**
- connection between the **pulmonary trunk** and **descending aorta**
- more common in **premature (Preterm) babies**. **(√)**
- May close spontaneously



Features

- left subclavicular thrill (sometimes **rough systolic murmur along the left sternal border**)
- **Continuous 'machinery' murmur (√)**
- large volume, bounding, collapsing pulse
- wide pulse pressure

Diagnosis → **Echocardiogram**

Management

- **Indomethacin / ibuprofen (NSAIDs) (ind=end=closes the duct)** (inhibits prostaglandin synthesis) closes the connection in the majority of cases. **(√)**
- If associated with another congenital heart defect amenable to surgery then **prostaglandin E1** is useful to **keep the duct open** until after surgical repair.

Indomethacin or **ibuprofen** → **closes** the duct.

Prostaglandin → keeps the duct **open**.

Key 65

14 days old baby is Cyanosed, Desaturated with Ejection systolic murmur.

→ **Tetralogy of Fallot**.

The cause of ejection systolic murmur here is → **pulmonary stenosis** (one of the four criteria of TOF).

Tetralogy of Fallot (TOF)

Tetralogy of Fallot (TOF) **is the most common cause of Cyanotic congenital heart disease**

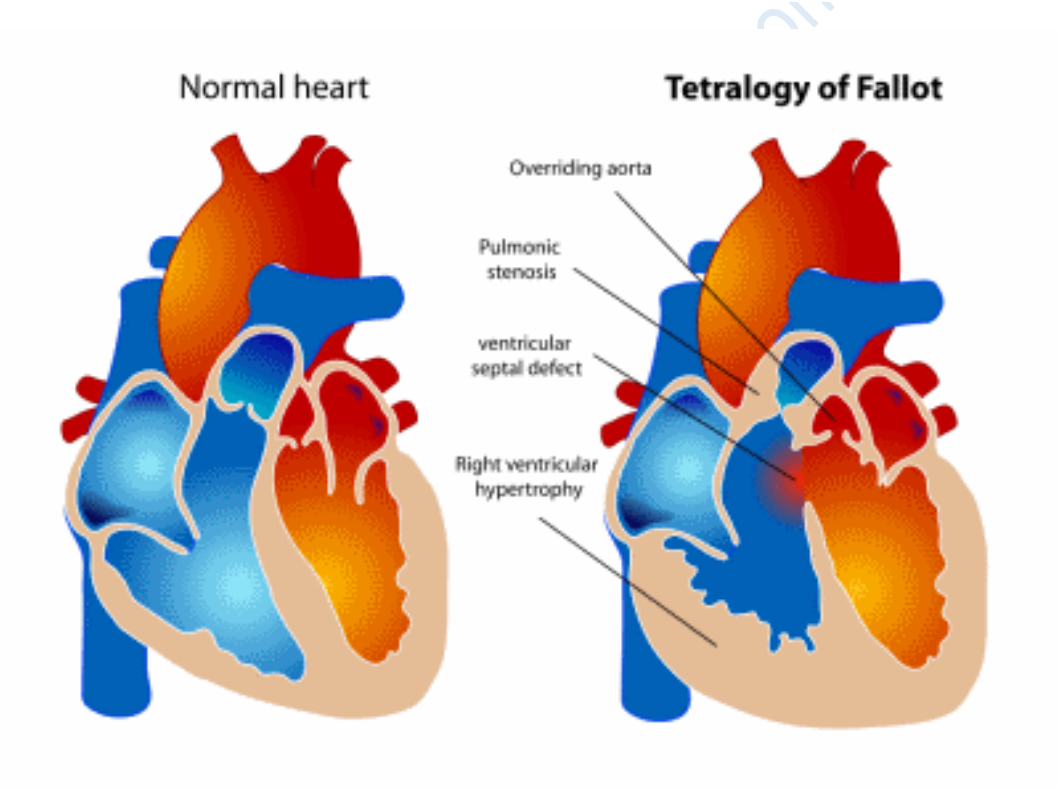
however, at birth, transposition of the great arteries is the more common lesion as patients with TOF generally present at around 1-2 months. *

It typically presents at around **1-2 months**, although may not be picked up until the baby is **6 months** old

TOF is a result of anterior malalignment of the aorticopulmonary septum.

The four characteristic features are:

1) Ventricular septal defect (VSD)
2) Right ventricular hypertrophy (RVH)
3) Right ventricular outflow tract obstruction "pulmonary stenosis" (PS) → **ejection systolic murmur**
4) Overriding aorta



The severity of the right ventricular outflow tract obstruction determines the degree of cyanosis and clinical severity.

Other features

- cyanosis
- causes a right-to-left shunt
- ejection systolic murmur due to pulmonary stenosis (the VSD doesn't usually cause a murmur)
- a right-sided aortic arch is seen in 25% of patients
- chest x-ray shows a 'boot-shaped' heart.
- ECG shows right ventricular hypertrophy

Management

- **Surgical repair** is often undertaken in two parts.
- Cyanotic episodes may be helped by **beta-blockers** to reduce infundibular spasm.

Key 66

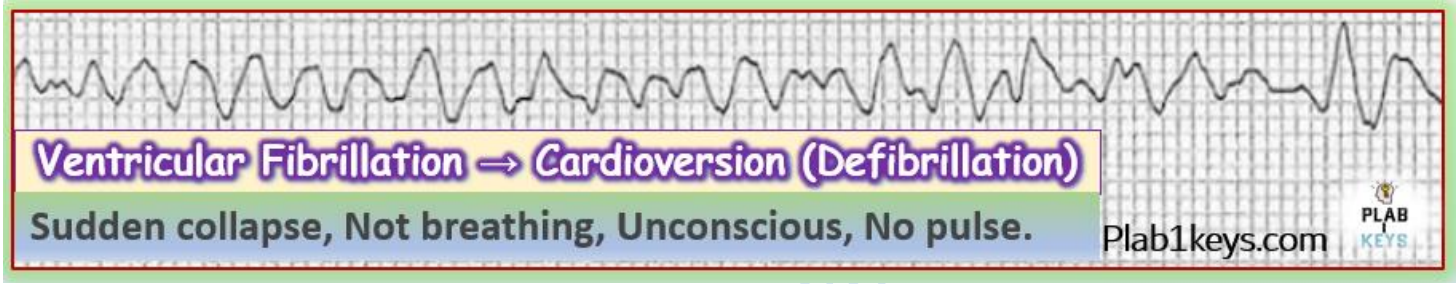
Familial Hypercholesterolemia

It is **Autosomal Dominant**

Q) When to highly suspect it?

1) **If Cholesterol is > 7.5 (Normal: <5 mmol/L)**

2**) Family History of "MI" in a first degree relative before the age of 60 or 2nd degree below 50.**

	First degree relatives: Parents and siblings. *Second degree relatives: Grandparents, aunts, uncles*
Key 67	**Ventricular Fibrillation** 
Key 68	**While in a hospital, an elderly patient was found unresponsive, no pulse and no breathing (No signs of life). Management?** → **Ring the emergency bell and call the resuscitation team. First** → **Start CPR 30:2.** → **Get Defibrillator.** → **Commence ALS when the resuscitation team arrives.** ***(In this order. If the first one was not given in the options, pick the 2nd one)***
Key 69	**Pan-Systolic murmur could be:**

	◙ **MR** → Mitral Regurgitation. ◙ **TR** → Tricuspid Regurgitation. ◙ **VSD** → Ventricular Septal Defect. **VSD (Ventricular Septal Defect):** ◙ **If small hole** → Asymptomatic ± Left sternal pan-systolic murmur with systolic thrill. ◙ **If large hole** → • Pan-systolic murmur along the left sternal border. • Left sternal heave, and systolic thrill. • Pulmonary HTN → Dyspnea, Fatigue. • May develop a right-to-left shunt → Cyanosis
Key 70	◙ **Myocardial Infarction** (Weak dead part of the cardiac muscle) can cause → **Congestive Heart Failure** → Backflow of the blood to the lungs → **Pulmonary Oedema**

	→ Desaturation, Dyspnea, Orthopnea, Crepitations → Perform **Chest X-Ray** to Diagnose → Perform **Echo** to identify the Underlying cause of the Pulmonary Oedema → Treat with **MONF** (**Morphine**, **Oxygen**, **Nitrates**, **Furosemide**)
Key 71	On ECG, if there is **no connection** between **P waves** and **QRS complexes** → **Complete heart Block** (**3rd degree** heart block).
Key 72	A 57 YO hypertensive patient is on Enalapril (ACE inhibitor) for hypertension developed annoying dry cough. What is the most appropriate action? → (Give **ARBs** e.g., **Losartan** instead -important-). Other **ARBs (Angiotensin II receptor blockers)** that could be the valid answer → **Losartan**, **Valsartan**, **Candesartan**. √ One of the important and common side effects of **ACE inhibitors** is **dry cough**. If developed, shift to **ARBs**. √ Another side effect of ACEi to remember is → **Hyperkalemia** (↑ serum K^+).

Key 73

Pansystolic murmur at the apex + Hx of Rheumatic fever

→ **Mitral Regurgitation** (MR).

Notes:

◙ MR can occur 2ry to MI (Rupture of papillary muscles).

◙ MR can occur 2ry to Rheumatic fever.

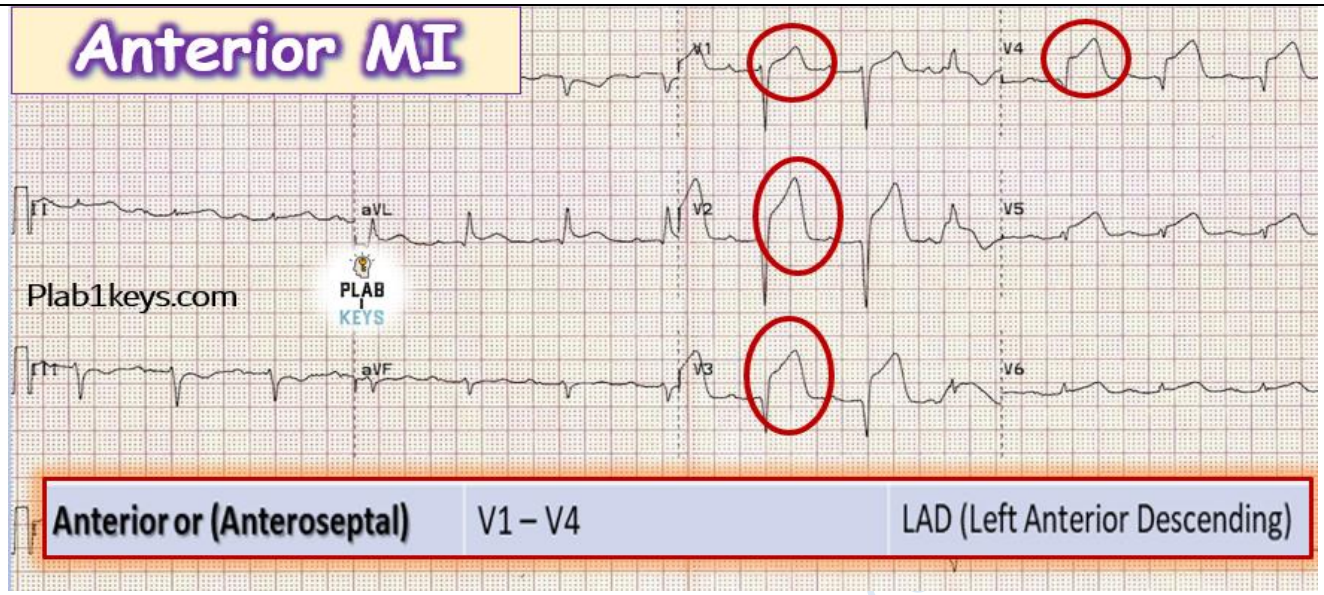
◙ MR may lead to → Right-sided (Congestive) heart failure (Ascites, LL oedema).

◙ MR may lead to → Pulmonary oedema

◙ Rheumatic fever can lead to either Mitral Regurgitation or Mitral Stenosis. We can decide based on clinical features.

Mitral Stenosis	Mid-Late Diastolic murmur, with opening click	At the Apex (left 5th ICS MCL)	Symptoms of Heart Failure
Mitral Regurgitation	Pan-Systolic murmur	At the Apex (left 5th ICS MCL), radiates to Axilla	Symptoms of congestive Heart Failure; edema, Ascites

Key 74	**Scenario** **A 60 Y/O patient with Hypertension, Previous MI and Asthma presents complaining of recurrent falls. He is on Salbutamol inhaler as needed, Aspirin, Corticosteroid inhaler (Beclomethasone), Indapamide, Enalapril and Amlodipine.** **The likely underlying cause of the recurrent falls** → **Postural Hypotension**. **Why?** → The patient is on multiple anti-hypertensive medications (CCB → Amlodipine ▌Thiazide-like diuretics → Indapamide ▌ACEi → Enalapril). These Blood Pressure Lowering agents are known to cause orthostatic "Postural" hypotension. **Management?** → **Blood Pressure Monitoring** + **Review the patient's medications**.

Key 75	
Key 76	An elderly patient fell and collapsed "syncope". He was transferred to the A&E and now he is fully conscious. ECG shows irregular rhythm. What is the next best **investigation**? → **Echocardiogram**. ◙ A **Holter ECG** (**24-hour ECG**) will not be beneficial as the ECG already shows Irregular Rhythm; hence, there is no point of using it again. ◙ **Echo** should be done to identify the **underlying cause** of this irregular rhythm so the treatment can be decided accordingly. ◙ The most common **Valvular heart disease** that causes **Syncopal attacks** is → **Aortic Stenosis** (ejection systolic murmur at the 2nd right ICS + Angina ± Dyspnea). ◙ Syncope during or shortly after exertion → Exercise ECG.

<table>
<tr>
<td></td>
<td>
This patient likely has AF causing TIA (syncope + irregular rhythm), and to determine the cause of his Atrial fibrillation, do Echo.

◙ Causes of AF:
√ Endocardium → Endocarditis, Mitral valve disease.
√ Myocardium → Cardiomyopathy.
√ Pericardium → Constrictive pericarditis.
√ HF, HTN, MI.
√ Hyperthyroidism, excessive alcohol intake, chronic lung disease.

<table>
<tr>
<td>Aortic Stenosis</td>
<td>Ejection Systolic murmur</td>
<td>Right 2nd ICS just lateral to sternum, radiates to Carotid artery</td>
<td>Dyspnea on activity, Anginal chest pain, syncope</td>
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<tr>
<td>Key 77</td>
<td>A 6-week-old baby presents with the features of progressive cyanosis, poor feeding and SOB since the age of two weeks. Holosystolic murmur is heard.

→ Tricuspid Atresia.</td>
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<tr>
<td>Key 78</td>
<td>- Preterm, continuous murmur → PDA</td>
</tr>
</table>

	- Cyanotic baby with ejection systolic murmur (due to pulmonary stenosis) → **TOF** (Tetralogy of Fallot). - Progressive (Severe) **C**yanosis + Poor feeding + Holosystolic murmur along the left sternal border → **Tricuspid Atresia**. - **A**cyanotic, Pan-systolic murmur → **VSD** (Others: Poor feeding and poorly gaining weight)
Key 79	**Before prescribing amiodarone** **Request → Serum Electrolytes and Urea** ***Amiodarone*** Amiodarone is a **class III antiarrhythmic** agent used in the treatment of atrial, nodal and ventricular tachycardias. The main mechanism of action is by **blocking potassium channels** which inhibits repolarisation and hence prolongs the action potential. **Monitoring of patients taking amiodarone** • **TFT** (Thyroid), **LFT** (Liver), **U&E** (Serum electrolytes and Urea), **CXR**, **ECG** **prior to treatment** • **TFT**, **LFT** every **6 months** • **ECG** every **12 months**

	Adverse effects of amiodarone use • Thyroid dysfunction: both hypothyroidism and hyperthyroidism • Corneal deposits • Pulmonary fibrosis (**The most serious**)/pneumonitis • Liver fibrosis/hepatitis • Peripheral neuropathy, myopathy • Photosensitivity • 'Slate-grey' appearance (Grey skin) • Thrombophlebitis and injection site reactions (So, usually given via central veins) • Bradycardia • Prolonged QT interval
Key 80	• A **racing** heart or **palpitation** is a common phenomenon in **Alcoholics** which is not serious or harmful. → **Reassurance**.

Key 81				
	Pulmonary Regurgitation	Early-Diastolic	Left 2nd ICS just lateral to sternum	Symptoms of Right-Sided Heart Failure

	After surgical correction of **Tetralogy of Fallot** early in life, the corrected **pulmonary stenosis** (one of the four criteria of **TOF**) can be complicated into **Pulmonary regurgitation** (diastolic murmur at the left upper sternal border) that can manifest many decades later. **A man who had cardiac surgery when he was a child presents with diastolic murmur.** Suspect → **pulmonary regurgitation** (on top of the corrected pulmonary stenosis as a child – due to TOF)
Key 82	**A patient who underwent surgery 2 days ago developed high fever, rigors, night sweats and systolic murmur.** New murmur + Fever → **Infective Endocarditis**. Investigation → **Blood Culture** then **Echocardiogram**.
Key 83	**Young Adult** + **Recurrent Palpitations** + **Light-headedness** + **Tachycardia**

	→ Think of SVT (**Paroxysmal Supraventricular Tachycardia**) = Narrow Complex. → **Valsalva manoeuvre** and **Carotid massage** → **IV Adenosine**: 6 mg → 12 mg → 12 mg → **Cardioversion** N.B. **Adenosine** is contraindicated in **asthmatics** → **Verapamil (CCB)** is the preferred option in SVT in a patient with **Asthma**.
Key 84	◙ **1st Degree** Heart Block and **Mobitz type** 1 usually → **do not require treatment** (as long as the patient is **Asymptomatic**). ◙ **Mobitz type 2** and Complete heart block (**3rd degree** heart block) → require **permanent pacemaker**. • **Initially → Atropine (first choice for all symptomatic bradycardia).** • **Then → Transcutaneous pacing. (Used to buy time until transvenous pacing is done).** • **Then → Transvenous pacing.** • **Then → Permanent pacing (Pacemaker).**

Key 85

Aortic Stenosis

- The commonest valvular disease in the **elderly** (Over 65).
- Usually **Asymptomatic** apart of **exercise intolerance (often mild)**.
- It can cause **syncopal fainting**.
- **Ejection systolic** murmur at the **right** 2nd ICS, louder on sitting **upright** and during **expiration**, and radiates to carotid.

A scenario of an elderly presents with mild exercise intolerance or is asymptomatic but visiting for the purpose of check-up is found to have ejection systolic murmur.

→ **Aortic Stenosis**.

The most appropriate investigation → **Echocardiogram**.

Key 86

Investigations following Syncope

Patients who have unannounced loss of postural tone leading to a period of unconsciousness need to be investigated for 4 main causes:

1) **Irregular rhythm** (**Cardiac syncopal events**): Usually abrupt, with rapid recovery and flushing → **12 Lead ECG.**

2) Low blood pressure or **postural drop** → frequent falls, dizziness while trying to stand from a sitting position, Hx of taking multiple medications

→ **Measure BP** while lying and 3 minutes after standing

(i.e. **Blood Pressure Monitoring**).

3) **Seizures** → need eyewitness to confirm, as there is usually post-ictal drowsiness or confusion.

4) **Hypoglycemia** → Hx of excessive alcohol intake, sweating, tachycardia, hypotension, confusion before falling → **Check blood glucose**

N.B. A vasovagal attack is usually due to overwarm environment, or prolonged standing period, or after visual stimuli e.g. seeing blood. The patients usually feel dizzy and ''tunnel vision'' before the attack.

	Another Clarification: **DDx of Sudden Falls.** • **Drop attacks** → Sudden falls **without** losing consciousness. • **Stokes Adam** → Unconscious + Abnormal ECG. • **Hypoglycemia** → Unconscious (or) ↓ level of consciousness + Sweating, do not recover unless given glucose. • **Vasovagal attacks** → Unconscious + Hx of prolonged standing, straining, pooping, heavy weight lifting or after visual stimuli e.g. seeing blood. The patients usually feel dizzy and ''tunnel vision'' before the attack. Usually in a YOUNG FEMALE (with NO chest pain, palpitation and with Normal ECG) • **Epilepsy** → Unconscious ± Post-seizure confusion
Key 87	**Digoxin Toxicity** • **GIT** (**Commonest**): **Nausea**, **Vomiting**, Anorexia. • Neurological: Hallucination, Confusion.

- **Visual**: **Yellow** green vision, (**Yellow haloes**), blurred vision.
- Arrhythmias: **Bradycardia**, V tach, Premature contractions.

Management:

- Order Digoxin level
- **Digibind [DigiFab] =** (digoxin immune FAB).
- Correct Arrhythmia
- Monitor Potassium

Aspirin Toxicity

√ The earliest symptoms of acute aspirin poisoning may include

→ **Ringing in the ears** **(tinnitus)** and impaired hearing.

√ More clinically significant signs and symptoms may include **rapid breathing** (hyperventilation), **vomiting**, **dehydration**, **fever**, **double vision**, and **feeling faint**.

Key 88

Thiazide-like diuretics

◙ Thiazide-like diuretics work by inhibiting sodium reabsorption at the beginning of the proximal part of the distal convoluted tubule (DCT) by blocking the thiazide-sensitive Na^+-Cl^- symporter.

◙ The main use of **bendroflumethiazide** was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as **indapamide** and **chlortalidone**.

◙ **Remember that: Furosemide** and **Bumetanide** (**Loop diuretics**) – inhibit the Na-K-Cl cotransporter in the **thick ascending** limb of the loop of Henle.

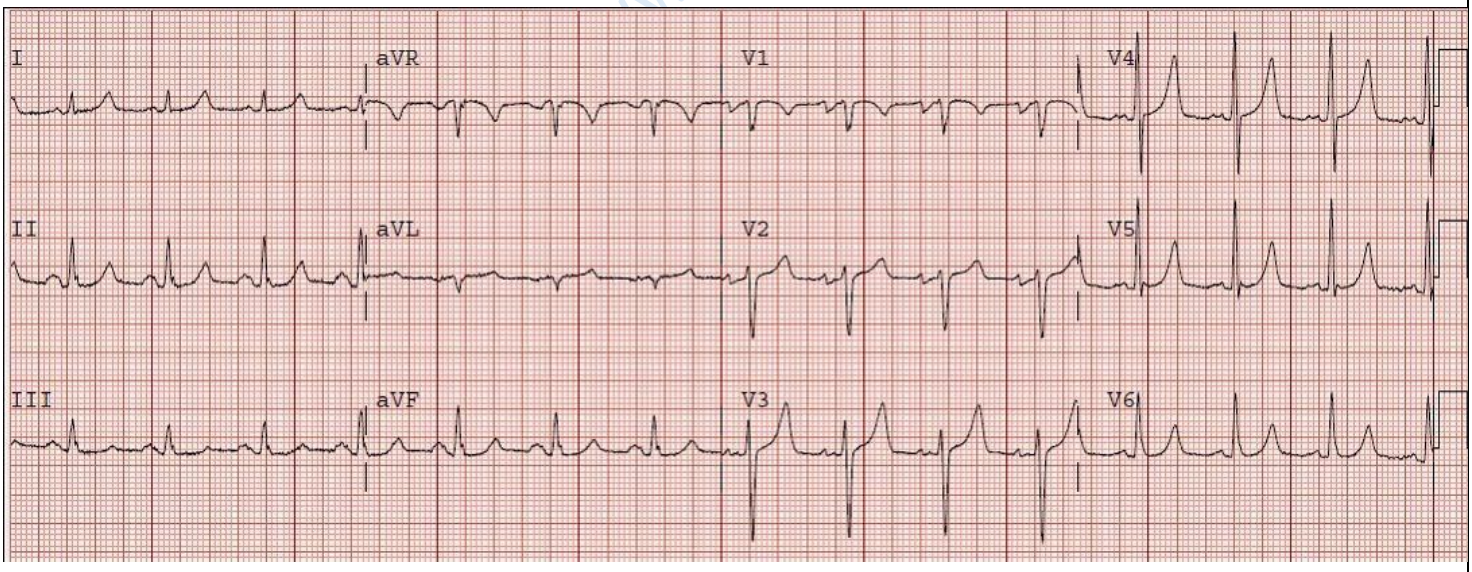
Common adverse effects

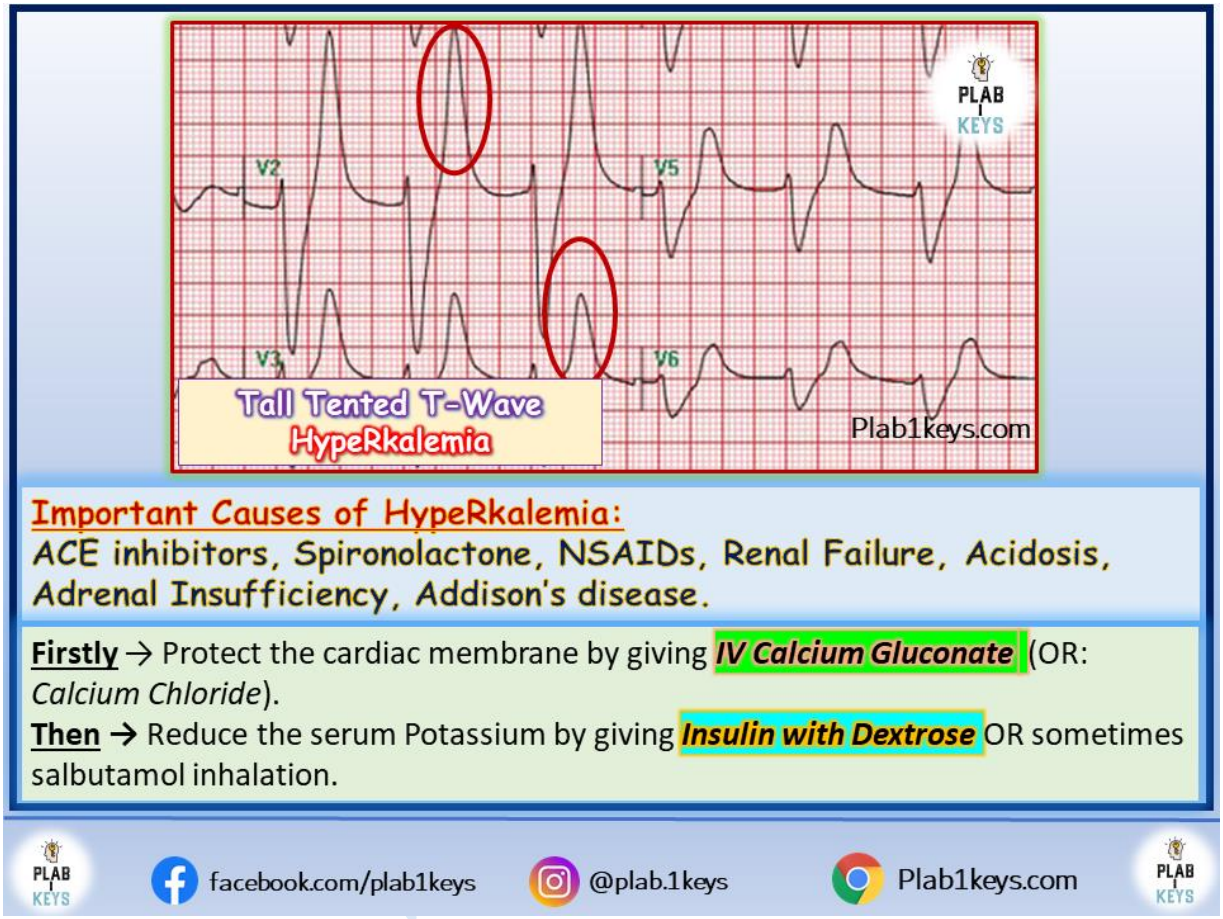
- **Postural Hypotension. √**
- **HypOkalemia and HypOnatremia. √**
- **Gout (Hyperuricemia). √**
- **dehydration**
- impaired glucose tolerance
- impotence
- Thiazide diuretics can cause **hypercalcaemia** and **hypocalciuria**

A patient with hypertension on treatment presents complaining of recurrent falls especially when trying to get up.

	→ **Postural hypotension** likely due to **thiazide** being used for hypertension.
Key 89	**A patient who is hypotensive (90/70), but is still conscious and the pulse is felt, his ECG shows a pattern between Ventricular tachycardia and Ventricular fibrillation. What is the treatment?** As the patient is hemodynamically unstable (Hypotensive) → **Cardioversion**. If there was No pulse (Hypotensive) → **Defibrillation**. **Remember:** Defibrillation = Asynchronized cardioversion **In 2 conditions:** √ Ventricular Fibrillation. √ Pulseless Ventricular Tachycardia.
Key 90	**2 weeks post MI, a patient was readmitted due to Hypotension, Tachycardia and Pulmonary edema. What is the likely underlying cause?** → **Acute Mitral Regurgitation**. **Acute mitral regurgitation (MR)**: "important √" pansystolic murmur

	◙ Occurs 2-15 days after the MI (Mostly inferior MI). ◙ Due to → Ischemia or rupture of the papillary muscles of the mitral valve. ◙ An early-to-mid systolic or **Pansystolic murmur** at cardiac apex is typically heard. ◙ May present with Hypotension, Tachycardia and Pulmonary edema (SOB and bibasilar crackles). ◙ Dx → **Echocardiogram**. ◙ Treatment → vasodilator therapy but often requires emergency surgical repair
Key 91	**Stable Angina**. √ In the case of stable Angina, the pain is precipitated by **predictable factors** such as **exercise and emotional stress**. This is because while exercising, the Oxygen demand is more than the Oxygen supply. √ Stable Angina is relieved by **Rest** and **GTN "Glyceryl Trinitrates"**. **Unstable Angina**

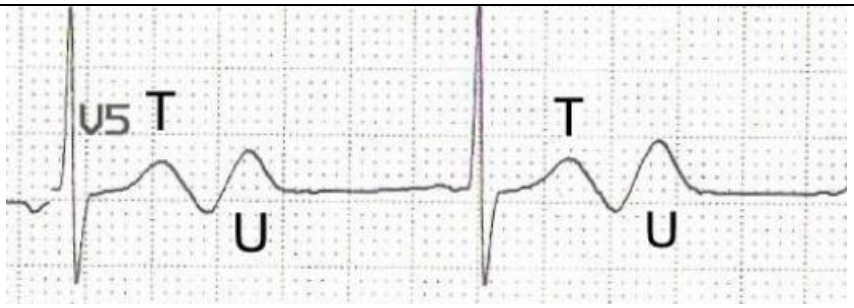
	Occurs mostly at rest, unpredictable, random. It is an emergency.
Key 92	◙ A baby with Progressive (Severe) Cyanosis + Poor feeding + Holosystolic murmur along the left sternal border → **Tricuspid Atresia** ◙ A baby who **does not** have cyanosis, presents with Poor feeding and poor weight gaining + Holosystolic murmur along the left sternal border → **VSD** Tricuspid Atresia → **Cyanotic.** VSD → **Acyanotic.**
Key 93	A patient known to have hypertension presents with Chest discomfort and Nausea. His ECG is as follows:  The ECG shows → **Tall Tented T-Waves** → **Hyperkalemia** (Likely 2ry to **ACE inhibitors** being used to control his Hypertension)



Remember that:

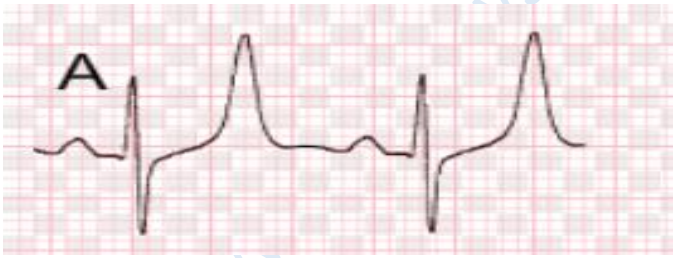
- **Spironolactone** and **ACE inhibitors** and **ARBs** → **HypeRkalemia**.
- **Loop diuretics**, **Thiazide diuretics** → HypOkalemia.

Key 94

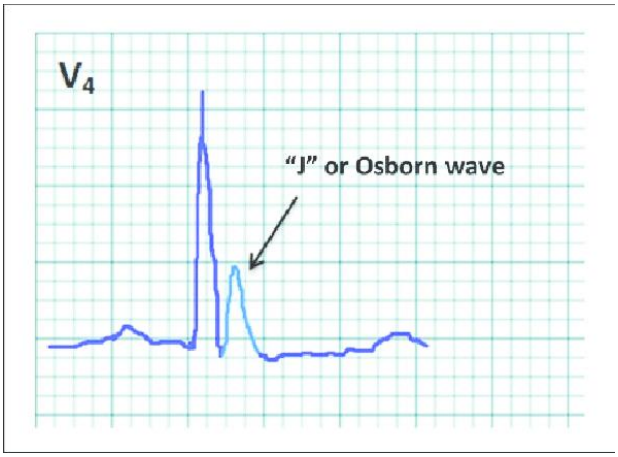


U wave in → Hypokalemia "HypUkalemia",

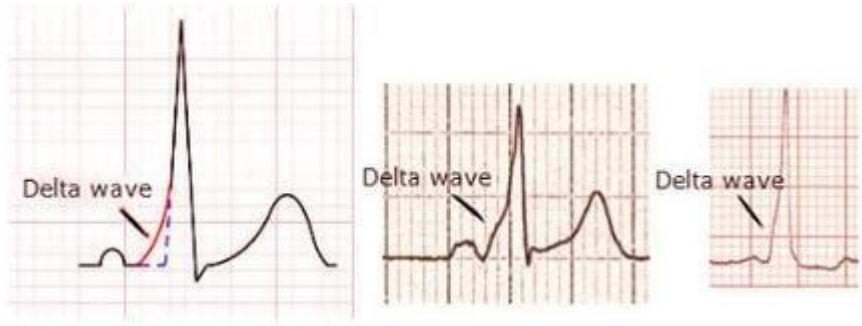
an additional wave after T wave.



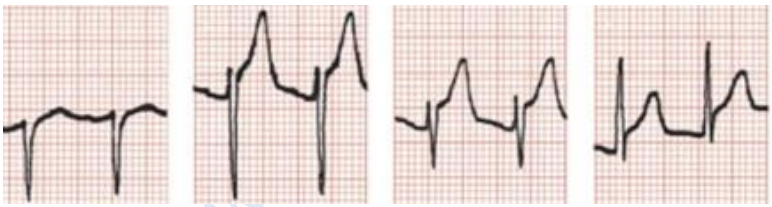
Tall Tented T-wave [Peaked T-wave] in → Hyperkalemia



J wave (Osborn wave) in → Hypothermia



Delta wave in → WPW syndrome (Wolff Parkinson White Syndrome)



Widespread Saddle Shaped ST Elevation with upward concavity

+ PR Depression in → Pericarditis.

Key 95

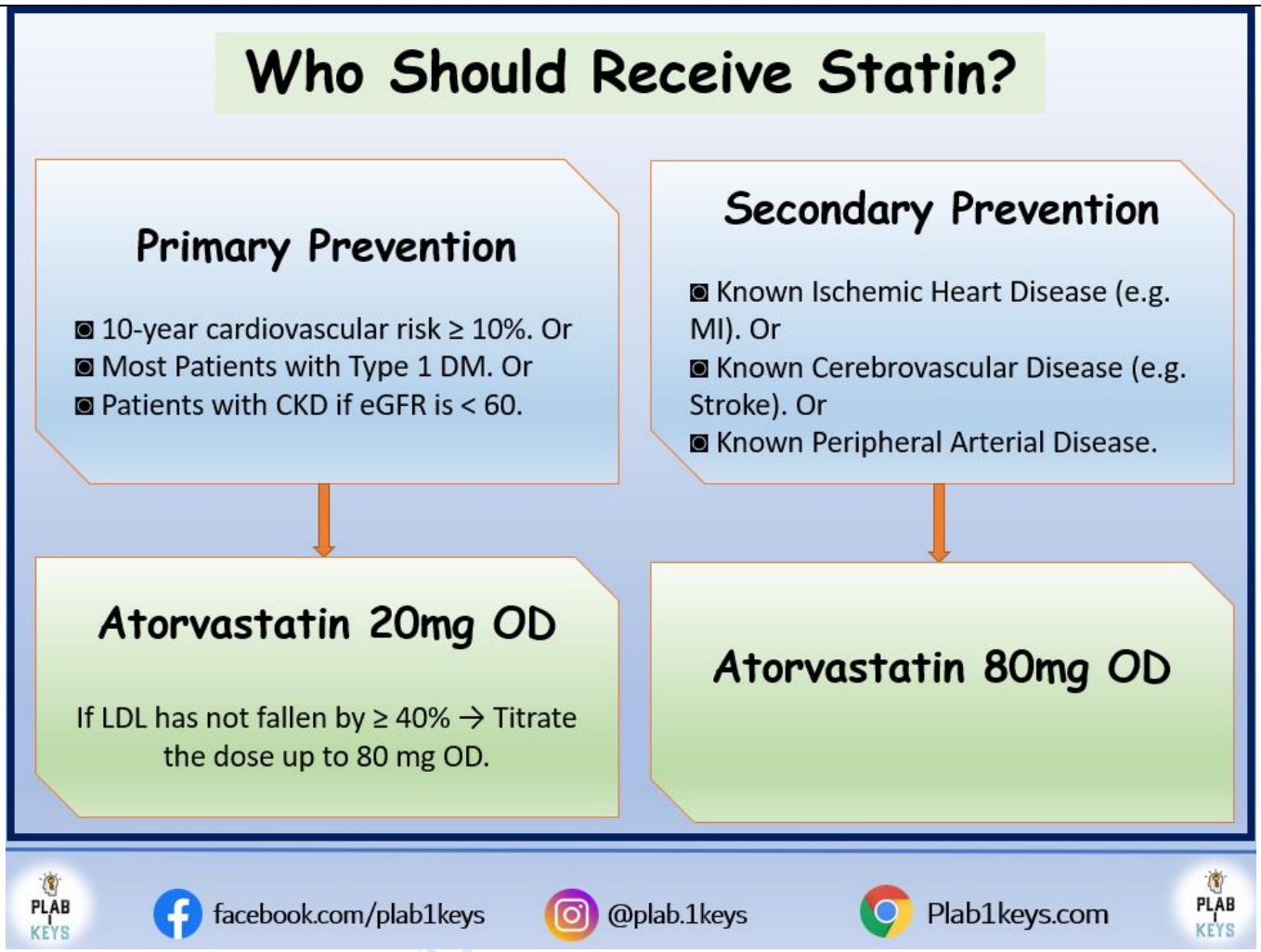
The table below summarises the most recent guidelines regarding antiplatelets:

Diagnosis	1st line
Acute coronary syndrome (medically treated) "MI"	Aspirin (lifelong) & Ticagrelor or clopidogrel (12 months)
Percutaneous coronary intervention (PCI)	Aspirin (lifelong) & prasugrel or ticagrelor (12 months)
TIA: Transient Ischemic Attack	Aspirin 300 mg **2 weeks** then Clopidogrel 75 mg (lifelong)
Ischaemic stroke	Aspirin 300 mg **2 weeks** then Clopidogrel 75 mg (lifelong)
Ischemic stroke + AF	Aspirin 300 mg for **2 weeks** then start **Anticoagulation** (e.g. Warfarin or DOAC- apixaban, rivaroxaban)
Peripheral arterial disease	Clopidogrel (lifelong)

Who should receive a statin?

- **All** people with **established cardiovascular disease** (**stroke**, **TIA**, **ischaemic heart disease**, **peripheral arterial disease**).

	• following the 2014 update, NICE recommend anyone with a **10-year cardiovascular risk ≥ 10% should receive statin.** • patients with **type 2 diabetes** mellitus should now be assessed using **QRISK2** like other patients are, to determine whether they should be started on statins. • patients with **type 1 diabetes** mellitus who were diagnosed more than 10 years ago OR are aged over 40 OR have established nephropathy. **Statins should be taken at night** as this is when the majority of cholesterol synthesis takes place. This is especially true for **simvastatin** which has a **shorter half-life** than other statins.

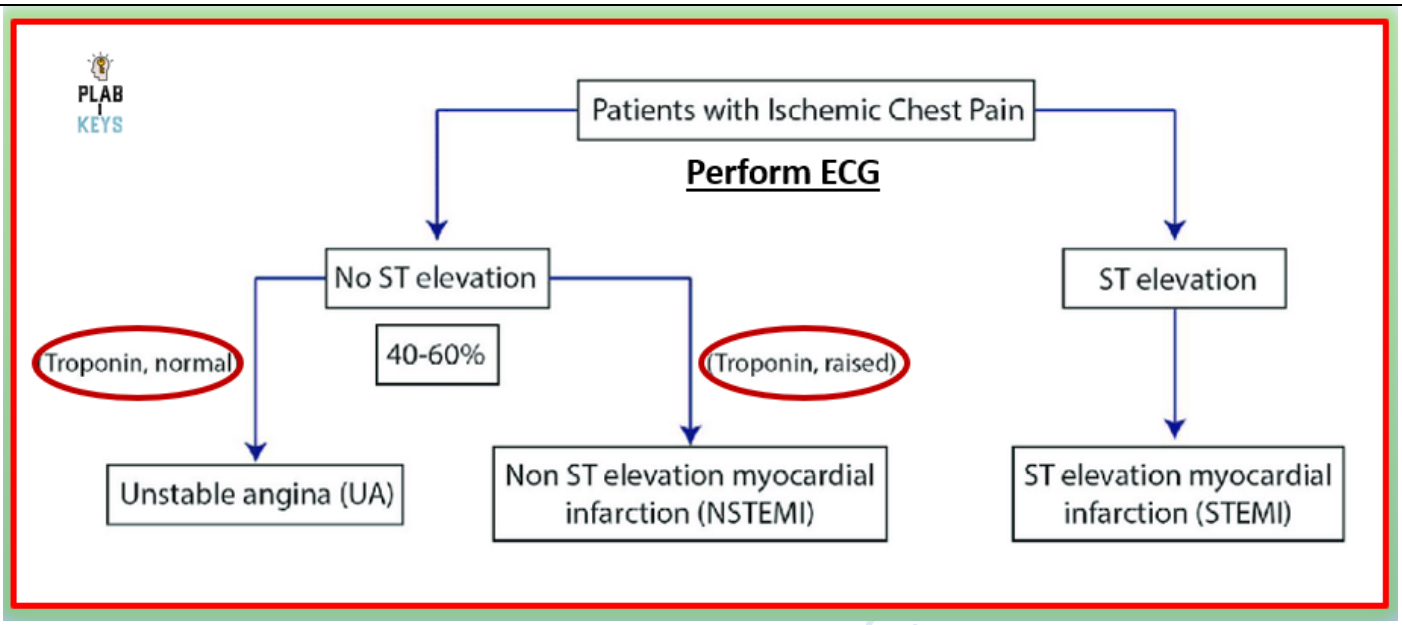


Key 96	Atrial fibrillation in an **unstable** patient → **Cardioversion** **If not in the options** → **IV amiodarone** (or IV flecainide) not oral! If **stable** → **BB**, (or CCB if he is asthmatic), (or digoxin if associated heart failure)

	Scenario, **A 70 YO female presents to the ED after a fall at home. She is confused, pale, with irregularly irregular pulse and cold peripheries. Her BP is 80/50 and HR is 150 bpm. ECG is done and shows narrow QRSs and absent P waves.** The **next step** in management is → **Immediate DC Cardioversion** ◘ The patient has **AF** (Irregularly irregular rhythm, Tachycardia, Absent P waves). ◘ Since she is **unstable** (Confusion, severe Hypotension) → **cardioversion**.
Key 97	**A man was hit by a car and sent to the ED. He is hypotensive with distended neck veins and faint heart sounds. His blood pressure is 82/47 and HR is 120.** The most appropriate management → [**Pericardiocentesis**]. **Cardiac Tamponade** **Accumulation of pericardial fluid under pressure** • **Beck's Triad**:

	Hypotension ▌**Muffled Heart Sounds** ▌**High JVP** (Distended neck veins). • **Dx**: **Echocardiogram** is diagnostic • **Rx**: **Urgent pericardiocentesis**.
Key 98	**Ventricular Fibrillation** — **Old, Sudden collapse, confusion, severe hypotension, Not breathing, Unconscious** → **Deliver immediate Shock** ''defibrillation''
Key 99	Atrial fibrillation in an **unstable** patient → Immediate shock If **stable** → BB, (or CCB if he is asthmatic), (or digoxin if associated heart failure) ***Scenario,*** A 70 YO female presents to the ED after a fall at home. She is confused, pale, with irregularly irregular pulse and cold peripheries. Her BP is 80/50 and HR is 150 bpm. ECG is done and shows narrow QRSs and absent P waves.

	The **next step** in management is → **Immediate DC Cardioversion** ◘ The patient has **AF** (Irregularly irregular rhythm, Tachycardia, Absent P waves). ◘ Since she is **unstable** (Confusion, severe Hypotension) → **cardioversion**. If cardioversion is not given, pick → **IV amiodarone**.
Key 100	**MI (Acute chest pain radiating to jaw, shoulder…) BUT without ST elevation on ECG. What to Do Next?** → Order Cardiac Enzymes, especially (**Troponin**) √ If Troponin is high → **NON-STEMI Elevation MI** √ Immediate management → **Give LMWH OR Fondaparinux** **√ recent exam + Aspirin 300 mg.**



Key 101

A patient's ECG shows SVT [Supraventricular Tachycardia].

→ **Adenosine**.

(Remember that Valsalva maneuverer and Carotid massage are tried initially and IV Adenosine is then given).

IMPORTANT NOTE:

If the patient is hemodynamically **unstable** (eg, hypotension, chest pain, shock, and or altered mental status)

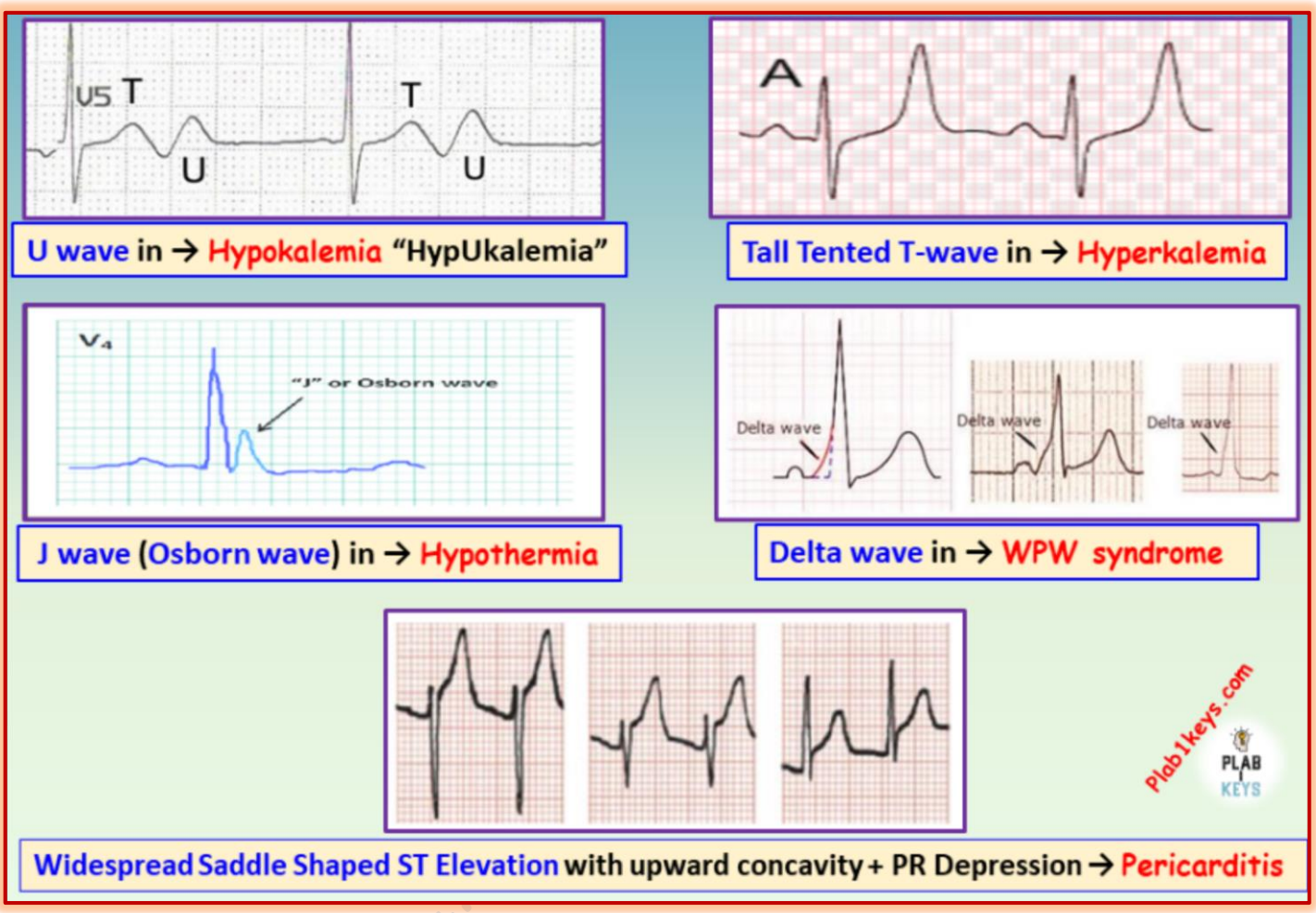
Proceed immediately to → electrical synchronised cardioversion.

Key 102

Important Side Effects of Diuretics

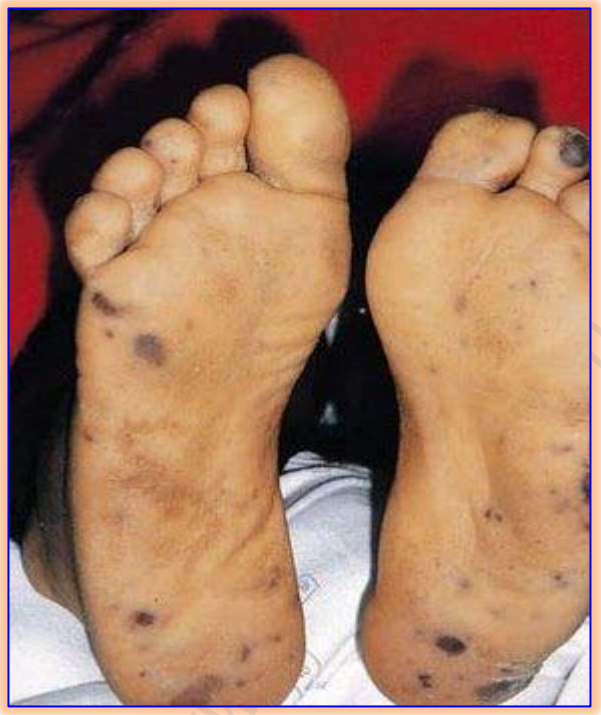
Loop diuretic	Thiazide-like diuretics	Potassium-sparing diuretics
e.g., Furosemide bumetanide	e.g., Bendroflumethiazide Indapamide	e.g., Spironolactone eplerenone
Hyponatremia	Hyponatremia	Hyponatremia
Hypokalemia	Hypokalemia	Hype**R**kalemia
Gout (hyperuricemia)	Gout (hyperuricemia)	Gynecomastia
	Postural Hypotension	
	Hyperglycemia (impaired glucose tolerance)	

Key 103



Key 104

A man presents with Fever, confusion, petechiae. This is a picture of his soles

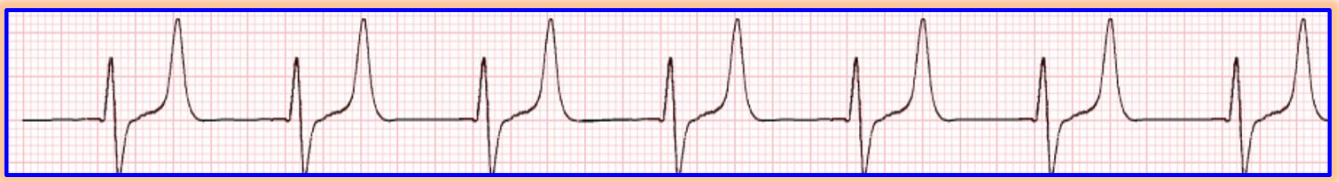


What is the most appropriate investigation?

→ **Blood Culture**

These lesions are likely **Janeway lesions** (minor criteria of infective endocarditis).

♦ Likely → **Infective endocarditis** → Do Blood culture **then** Echocardiogram.

Key 105	**The initial step for a patient with the following ECG:**  initial line → give **Calcium Chloride (or Calcium Gluconate)** **(or Calcium carbonate)** (to prevent cardiac arrhythmia). This is likely a case of **Hyperkalemia** (**Tall Tented T waves** are seen on the ECG). --- **Note**, If severe hyperkalemia (> 6.5), and there is no hyperkalemic ECG changes, we still give Calcium carbonate as a next step to protect the heart.
Key 106	**A patient is taking several drugs including Ace inhibitors + diuretics and other drugs. Then, He developed Hyperkalemia.** The initial step → **Withhold (stop) ACE inhibitors**.

	Remember that ACE inhibitors can cause hyperkalemia. One of the initial steps of the management is to stop the cause.
Key 107	◙ **An elderly female with a history of Atrial Fibrillation presents to the A&E with speech disturbance and asymmetric weakness of face and arm. These symptoms started 3 hours ago. CT scan of the head shows no hemorrhage. The "long-term" management of this patient would involve:** → **Rivaroxaban** (or any other DOAC e.g. **Apixaban**, **Rivaroxaban**, **Edoxaban, Dabigatran**) "Now preferred over Warfarin" This patient with TIA/ ischemic stroke will need to take Aspirin 300 mg for 2 weeks, then rivaroxaban (DOAC) and atorvastatin 80 mg for long-term management. If he did not have AF, clopidogrel will be given instead of DOAC. --- ◙ **In patients with Hx of ischemic stroke, the <u>long-term</u> medications:** √ **with** Atrial Fibrillation → DOAC (eg, apixaban) + Statins (eg, Atorvastatin). √ **Without** Atrial Fibrillation → Clopidogrel + Statins (eg, Atorvastatin). **Important: What if a patient with a history of stroke is already on warfarin and presents with a new onset atrial fibrillation?** Since he is already on an anticoagulant, he **should continue on warfarin** and should **not** be switched to DOAC (eg, Edoxaban) unless his INR is increased.

2ry Prevention (Long-term management) of Ischemic Stroke/ TIA:

[To prevent further stroke in the future].

√ Control Blood Pressure.

Remember, if he has **DM**, pick or add ACEi eg, ramipril.

√ Statins (for **All** patients regardless of their cholesterol baseline level).

√ Ani-platelets (or) Anti-coagulation: (**Based on presence or absence of AF**):

• If there is **Atrial Fibrillation** → Anticoagulants: **Warfarin [or] DOAC** (**Dabigatran/ Apixaban/ Rivaroxaban/ Edoxaban**). DOAC is now preferred.

(Warfarin is almost obsolete nowadays. DOAC is recommended instead)

• If **No Atrial Fibrillation** → Antiplatelets: **Clopidogrel** 75 mg OD.

Important: What if a patient with a history of stroke is already on warfarin and presents with a new onset atrial fibrillation?

Since he is already on an anticoagulant, he **should continue on warfarin** and should **not** be switched to DOAC (eg, Edoxaban) unless his INR is increased.

Key 108	**A patient has recovered form TIA. What score is helpful to determine the risk of a stroke in the following week?** → **ABCD2 Score**. ◙ The **ABCD2 score** (Prognostic) is used to **identify the risk of future stroke in patients who have had a suspected TIA in the following 7 days**. **√ IMPORTANT: ABCD2 score is Not advised to be used now according to the recent 2019 CKS guidelines. Therefore, it will not be a valid answer anymore. Thus, this question is to not be considered. We have added it just in case!** ♦ The **CHA2DS2-VASc score** is used to determine **the need to anticoagulants** in a patient who has **atrial fibrillation**.
Key 109	**A Diabetic patient with heart failure on beta-blockers, ACE inhibitors, insulin and furosemide was found to have hypokalemia. What is the likely cause?** → **Furosemide**. (Loop diuretics such as furosemide can cause hypokalemia).

	HypOkalemia	HypeRkalemia
	• **Loop Diuretics** (e.g. Furosemide) • **Thiazide-like diuretics** (e.g. bendroflumethiazide, indapamide) • **Vomiting** and **Diarrhea** • **Villous Adenoma** • **Renal tubular failure** • **Cushing Syndrome** • **Conn's disease** (1ry hyperaldosteronism)	• **ACE inhibitors**. • **Potassium-sparing diuretics** (e.g. Spironolactone/ Eplerenone) • **CKD "Chronic Kidney Disease"**. • **Addison's** (1ry Adrenal Insufficiency). • **Congenital Adrenal Hyperplasia (CAH)**.

Key 110

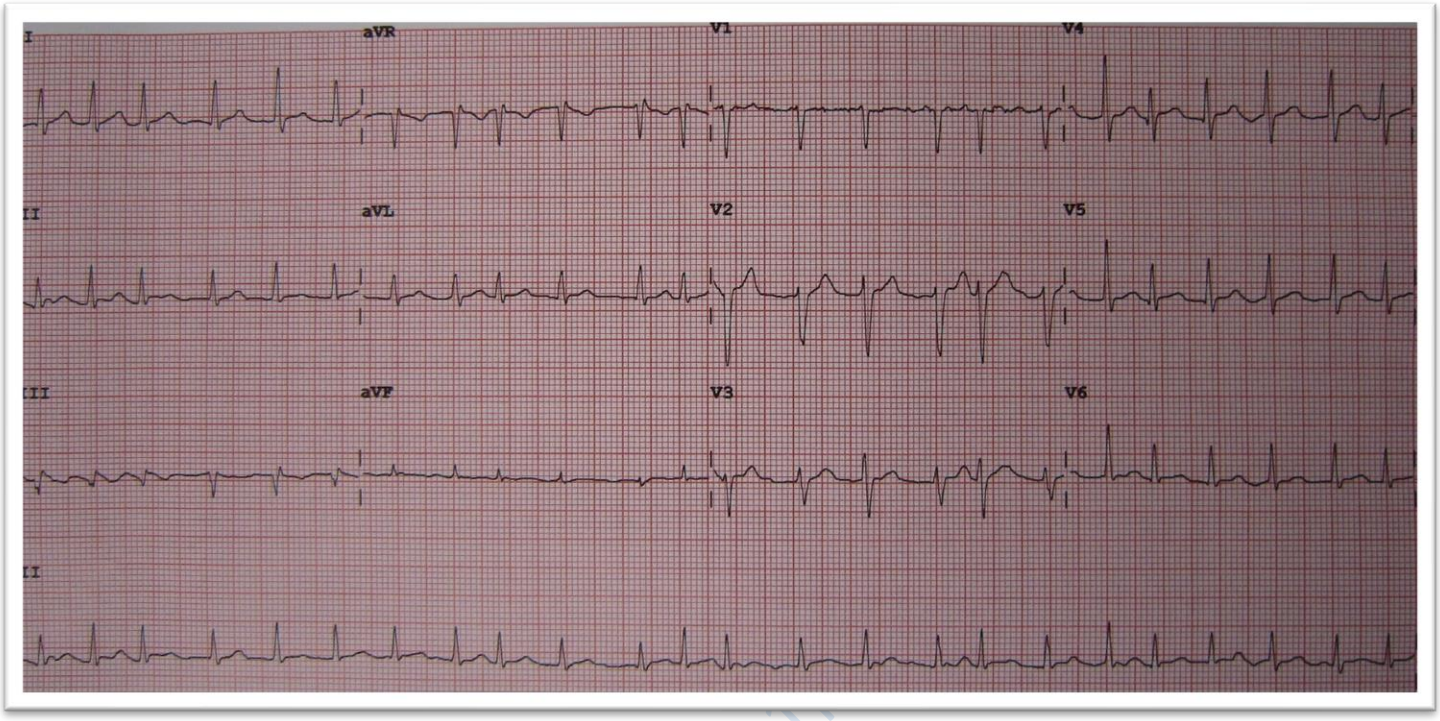
A patient with hypertension on treatment presents complaining of ankle swelling/ edema. The likely cause of this ankle oedema is:

→ **Amlodipine** (a calcium channel Blocker).

√ This patient is likely taking CCB for his HTN.

√ CCB can cause ankle swelling as it causes vasodilatation.

	2 Important Side effects of Calcium Channel Blockers (e.g. Diltiazem, Amlodipine) to be remembered: ◙ **Ankle Swelling / oedema** ◙ **Gingival Hyperplasia** So, for one who takes CCB such as ***diltiazem***, ***amlodipine***, ***verapamil***, ***nifedipine***, he might get **swelling of his → Ankle + Gingiva.**
Key 111	**Before prescribing amiodarone, what investigation should be ordered?** → Serum **Electrolytes** and Urea. **(Do not mix thing up):** The 2 most important tests to be done before initiating **lithium** are: ♣ (**TFTs**) Thyroid Function Tests, and: ♣ (**KFTs**) Kidney Function Tests.
Key 112	**The following ECG is done for a 58 YO man who presents with palpitation. He is otherwise healthy. What is the most appropriate line in Rx?**



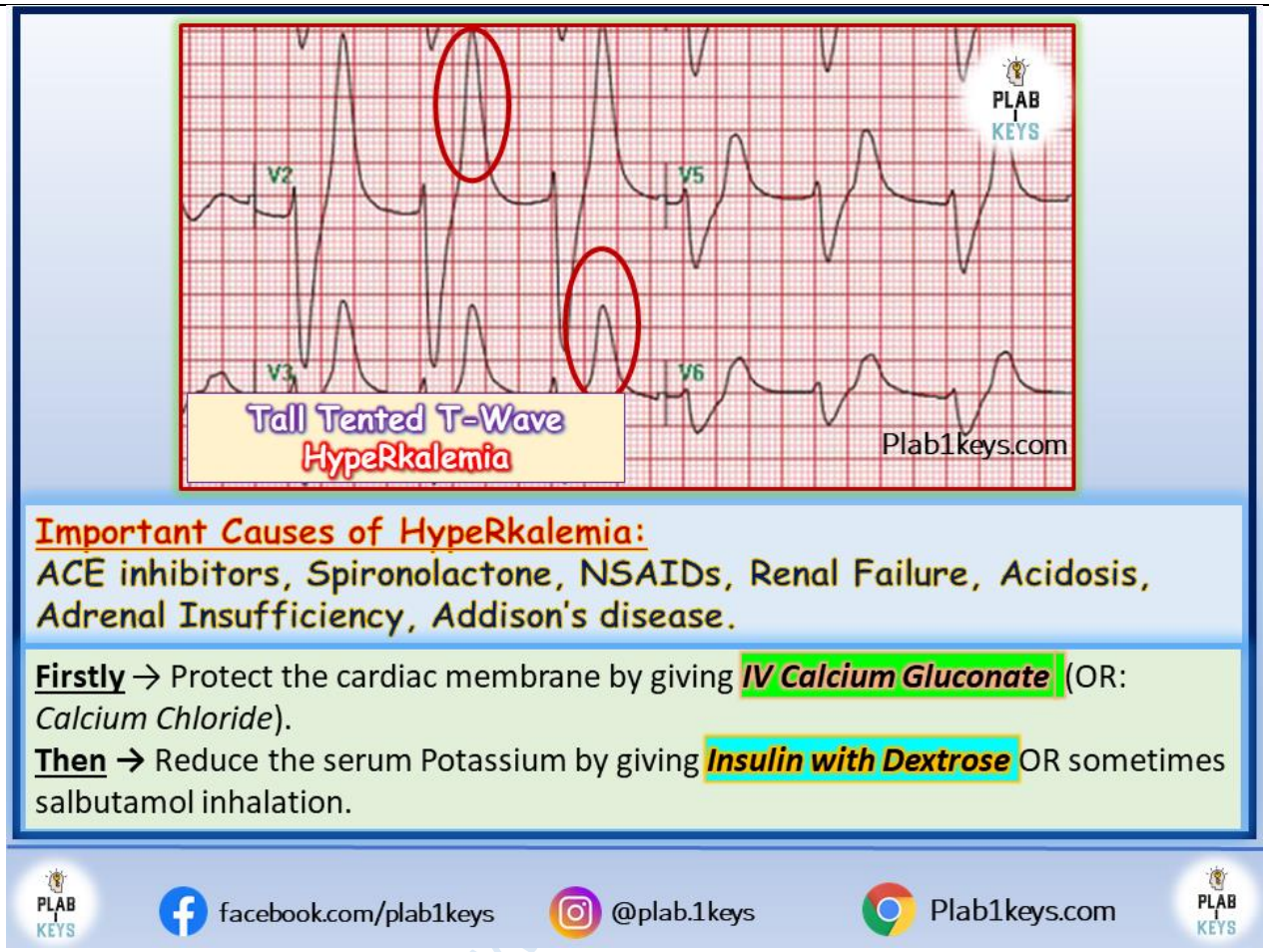
→ **Beta-blockers (eg, metoprolol, atenolol)**

◙ Agents used to control rate (**Rate Control**) in patients with **Atrial Fibrillation** **(AF):**

- **Beta-blockers** (eg, atenolol 50-100 mg PO OD, bisoprolol, metoprolol) → First line but Contraindicated in Asthma.
- **Calcium channel blockers [non-dihydropyridine = rate-limiting CCB]** (eg, diltiazem, verapamil) → if **Asthmatic** patient.

	○ **Digoxin** → (not considered first-line anymore as they are less effective at controlling the heart rate during exercise. However, they are the preferred choice if the patient has **coexistent heart failure**). √ **If haemodynamically unstable** (eg, SBP ≤ 90) → **Cardioversion** (Shock). √ If the patient with AF is **unstable** (eg, hypotension) and the AF has **just started** and no cardioversion in the options, Pick → **IV amiodarone**. **Imp √**
Key 113	**A patient presented with chest pain and breathlessness. Pulse rate is 35 b/m. ECG shows broad complexes with atrioventricular dissociation. Most appropriate initial treatment?** A. Adenosine B. carotid massage C. **atropine** D. verapamil E. Amiodarone ◙ The **first drug of choice** for **Symptomatic Bradycardia** (Dizziness, feeling unwell) → **Atropine** (Given 0.5 mg IV push and may be repeated up to a total dose of 3 mg).

Key 114	**A 76-year-old man was found outside by his carers this morning. He doesn't remember what happened but denies history of pain. Temperature 35.1, BP: 102/70mmHg, PR: 108bpm, mucous membrane is dry. No stiffness of any limb, his heart sound is normal. His chest is grossly normal apart from some scattered coarse crackle in his Left Lower lung zone. He was catheterized and urinalysis showed Blood+++, Protein ++ and Ketone +, ECG showed peaked T wave and broad complex Tachycardia. Which of the following is the appropriate initial intravenous medication he should have?** A. Amiodarone B. **Calcium gluconate** C. Co- Amoxiclav D. Insulin- Glucose infusion E. Sodium Bicarbonate **Peaked T** wave suggesting **Hyperkalemia** (2ry to kidney injury in this case). → **IV calcium gluconate** (or calcium chloride) to protect the heart should be given initially.



Key 115

A question about a patient with unstable HR > 150, BP 80/60 and Having broad complex tachycardia. Most appropriate management was asked?

→ **DC Cardioversion**.

Patient is **unstable**, **SBP < 90** → (Cardioversion) Shock.

- Broad complex tachycardia + low BP (unstable) ⟶ **Cardioversion**
- Pulseless V. Tach or V. Fibrillation → Immediate **Defibrillation**.

Key 116	**A 71-year-old man with 3 weeks history of fever, 1 month post inferior myocardial infarct, chest pain with soft systolic murmur, inverted Q waves in leads I, II & aVF. Temp- 37.5, BP- ?118/68** A. Pericarditis B. Costochondritis C. Pulmonary Embolism D. **Infective endocarditis** E. Papillary muscle rupture **New Murmur** + **Fever** → think of **Infective Endocarditis (IE)** ± Malaise, Rigors, Fever The initial step → **Blood Culture**. Then → **Echo**
Key 117	**Patient with Hx of MI presented after a few days with chest pain which aggravates on inspiration and is relieved on bending forward. Most likely Diagnosis?** A. **Pericarditis** B. Pulmonary Embolism C. Pleural Effusion

	◙ **Pericarditis** (A Complication of MI that can develop shortly after the MI within 2 days) and **Dressler's syndrome** (presents 2-6 weeks after MI) both have t'e same features → **Pleuritic chest pain that worsens on lying flat and during inspiration**, Pericardial rub, Widespread Saddle shaped ST elevation on the ECG. ◙ They can also lead to Pericardial effusion (Enlarged globular heart on chest X-ray) and if severe enough, Cardiac Tamponade can also develop (also enlarged globular heart on the X-ray). ◙ **Management of Pericarditis (imp):** √ A full-dose **NSAID** should be used (eg, aspirin, 2-4 g/d; **ibuprofen** 1200-1800 mg/d; indomethacin 75-150 mg/d); treatment should last at least 7-14 days. √ **Colchicine** (as an adjunct to NSAIDs to ↓ inflammation and recurrence).
Key 118	**A 60 YO man with Hx of smoking, HTN and DM presents to his GP complaining of 25 minutes left side dull aching chest pain radiating to his jaw. He was given Aspirin 300 mg by his GP and then sent to medical services in a local hospital. He is no longer in pain. The ECG is normal. The troIonin is elevated 202 ng/L (Normal: < 5 ng/L). What is the next step in management?** E) Alteplase. F) **Subcutaneous fondaparinux**. G) IV Glyceryl trinitrate (GTN).

H) IV Morphine.

Since the ECG is normal, alteplase is wrong.

Since **ECG is normal** and **Troponin is high** → **Non-STEMI**

→ **Anti-coagulation** (**LMWH** e.g. **Dalteparin**, **Enoxaparin** or **Fondaparinux**).

+ **Oral Aspirin 300 mg**.

Key 119

Points on Alcohol and Heart Disease

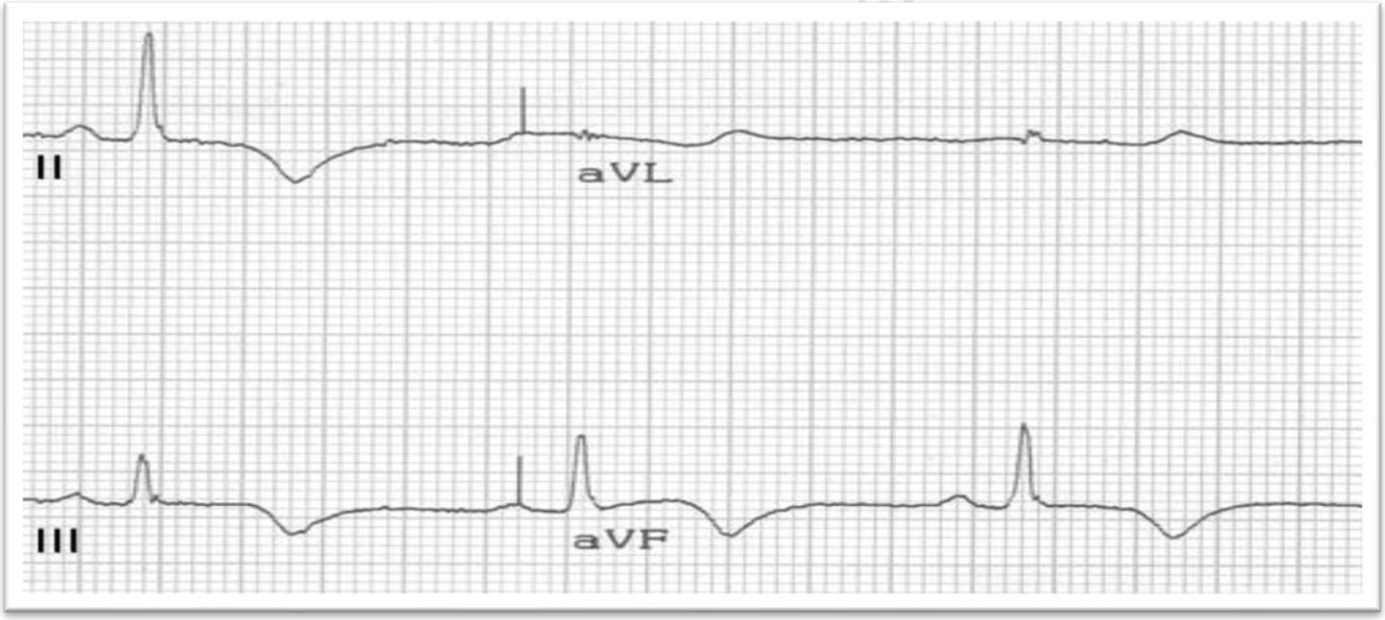
√ **Ankle swelling** and **orthopnea** → features of **Heart Failure**.

√ Excessive alcoholism can lead to → Alcoholic Cardiomyopathy (Cardiac Enlargement on Chest X-ray) → which can cause Atrial Fibrillation.

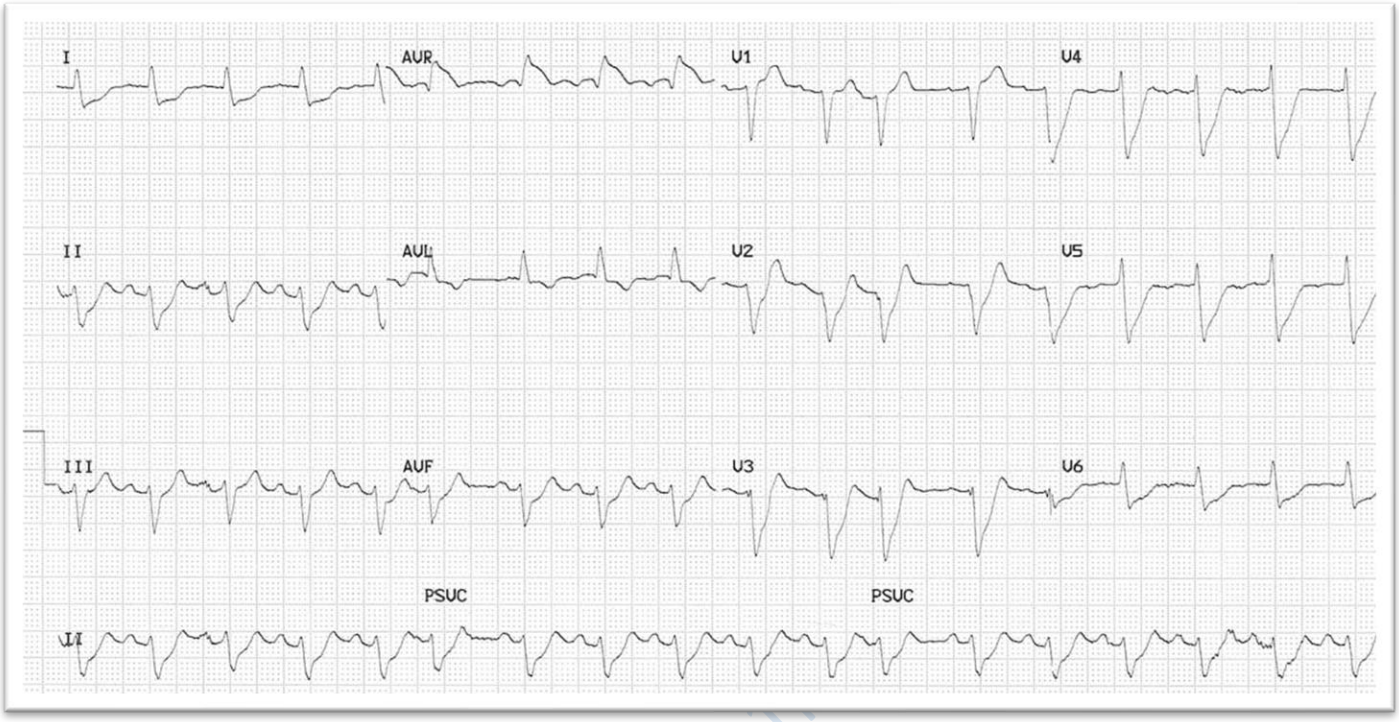
- The most common type of arrhythmia that develops in patients with **alcoholic cardiomyopathy** is → **Atrial Fibrillation**.

√ Acute alcohol intake can lead to → AF or flutter ((Holiday heart Syndrome)).

√ AF presentation: Palpitation, Dyspnea, Dizziness or Syncope, Chest

	discomfort or pain, Stroke or TIC, Irregularly irregular pulse.
Key 120	**A 59 YO hypertensive patient presents to the A&E complaining of dull central chest pain for around 4 hours. His vitals are as follows:** **HR: 99, BP: 155/95, RR: 21, O2 sat on room air: 97%** **Chest X-ray is normal. Troponin level was sent and still pending.** **He was given IV morphine for his chest pain.** **The ECG is as follows:**  **What is the most appropriate next step in management?** Chest pain + T wave inversion suggests → myocardial ischemia. In this case, 2 drugs should be given immediately:

	√ Oral **Aspirin 300 mg**. √ SC **LMWH or Fondaparinux**. Pick the one that is given in the choices. "low-risk patients can be treated **conservatively**. However, if subsequent ischemia develops → **coronary angiography** with PCI". What if the ECG shows features of left main coronary artery occlusion (Wide spread ST depression + ST elevation in aVR)? → Emergency coronary angiography.
Key 121	**A 61 YO patient presents to the A&E complaining of dull central chest pain for around 4 hours. His vitals are as follows:** **HR: 75, BP: 135/85, RR: 21, O2 sat. on room air: 97% He was given IV morphine for his chest pain. The ECG is as follows:**



What is the most appropriate next step in management?

This ECG shows the typical features of **Left main coronary artery occlusion**:

- Widespread ST depression, and
- ST elevation in aVR.

Thus → **Emergency coronary angiography**.

To determine which artery is occluded so we can plan for interventional or surgical procedures.

Key 122	**Remember:** **Long-term antiplatelet/anticoagulant drugs in:** **√ Ischemic stroke (or TIA) → Aspirin for 2 weeks, then clopidogrel for life.** **√ Ischemic stroke + AF → Aspirin for 2 weeks, then warfarin/DOAC for life.** If he was already on warfarin (anticoagulant) and develops atrial fibrillation (AF), continue on warfarin and do not shift to DOAC unless his INR is poor. **√ MI → Aspirin for life, clopidogrel (or ticagrelor) for 12 months.**
Key 123	**AF + Stable patient** **→ BB (eg, metoprolol) or CCB (if asthmatic) or Digoxin (if with HF)** **AF + Unstable patient (e.g. SBP ≤ 90, HR > 150, Loss of conscious)** **→ Cardioversion** **AF + Unstable patient but presents > 48 hours after beginning of symptoms** **→ Do not cardiovert; but give rate control (e.g. BB) + LMWH** **→ Then: assess for long-term intake of Warfarin or DOAC (e.g. apixaban, rivaroxaban, dabigatran) upon discharge using CHA2Ds2-VASc score.**

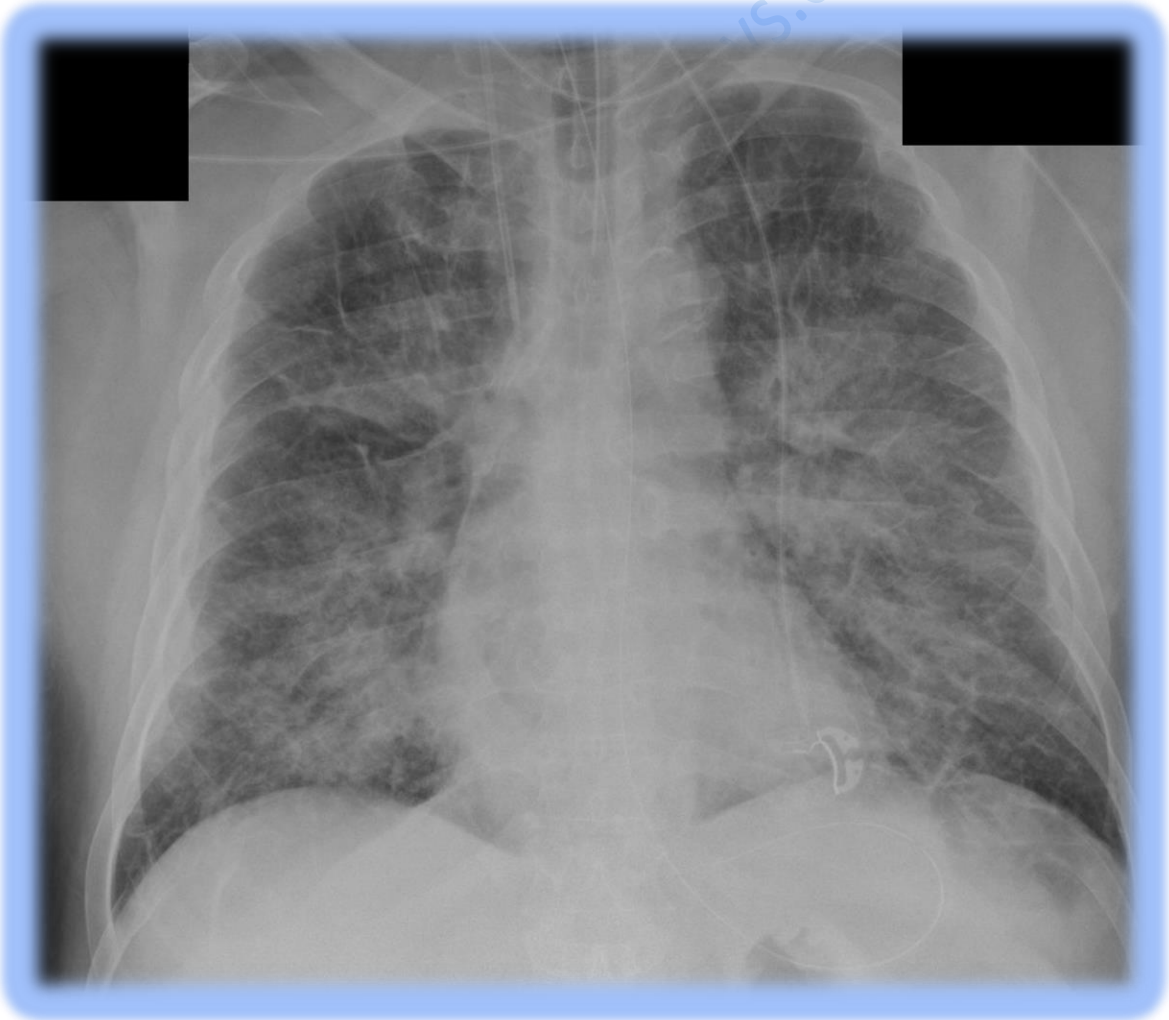
Key 124

A 60 YO asthmatic man presents to A&E complaining of chest discomfort and difficulty in breathing over the past few hours. On chest auscultation, there are widespread fine inspiratory crepitations. His vitals are as follows: HR 100, RR 28, BP 120/80, O2 saturation 90%.

Chest X-ray was performed (see the picture below). ECG: normal. Echo showed left ventricular impairment.

The patient was given high flow O2, sublingual glyceryl trinitrates, and IV furosemide (Lasix). His breathing improved significantly.

What is the most appropriate medication to be added?



This patient has acute pulmonary edema 2ry to left ventricular failure.

Pulmonary edema:

Kerley B lines, **bat's wing hilar shadow**, **crepitations**, **SOB**, **low O2 sat**.

Echo in the stem shows **left ventricular impairment (HF).**

HF can lead to → Pulmonary edema

Remember,

Management of HF:

- **Furosemide** (for symptomatic relief) "was already given here".
- Start with either **Beta-blocker** or **ACE inhibitor** (one medication at a time).
- If still? Add the other medication.
- If still? Add spironolactone

Since this patient is **Asthmatic**, we would begin with ACE inhibitors (e.g. lisinopril) instead of BB.

BB is better to be avoided in asthma as it can cause bronchospasm and thus aggravates asthma.

	So, the answer is that this patient should be discharged on → **ACE inhibitor (e.g. Lisinopril)**.
Key 125	**A 50 YO man with Hx of MI six months ago presents with a 3-day history of fever, SOB and chest pain. He looks moderately ill. His temp, is 38.2 and his BP is 140/77. On auscultation, a holosystolic murmur over the apex is heard. His ECG shows Q waves in leads I and aVL. What is the most likely Dx?** √ Do not rush and pick papillary muscle rupture thinking it is mitral regurgitation (evidenced by the holosystolic murmur over the apex)! If MR, why there is fever? √ Note that papillary muscle rupture usually occurs 2-15 days after MI, not 6 months! √ Also, papillary muscle rupture is more common with inferior MI (II, III, aVR). √ Q waves here → permanent markers of necrosis (indicate previous MI), here, likely lateral MI (I, aVL, V5, V6). **So, what is the correct answer??** **Remember**:

	FEVER + NEW MURMUR → INFECTIVE ENDOCARDITIS!
Key 126	**An old patient with triple vessel disease presents with sudden onset chest pain of 4 hours, shortness of breath, dizziness and sweating. His ECG shows ST depression "ischemia" in several leads. His blood pressure is 140/80. The patient is anaemic with haemoglobin level of 62 g/L. What is the most appropriate management?** → Dual antiplatelets (Aspirin + Clopidogrel) + Fondaparinux + Blood transfusion. **Notes:** √ Triple vessel disease means that 3 big vessels (the left anterior descending, right coronary and circumflex arteries) have blockages from atherosclerotic plaques. √ This patient has ACS "acute coronary syndrome" secondary to anemia and the pre-existing triple vessel disease.

	√ Aspirin (oral) and fondaparinux (SC LMWH) are given whenever there is heart ischemia. **√ Blood Transfusion is indicated if:** ♠ **Hb < 80 g/L** + **Symptoms of Anemia**. Or: ♠ **HB < 70 g/L** + With or Without **Symptoms of Anemia**.
Key 127	**Elderly + Episodes of Fainting + SOB + Systolic murmur at the right second intercostal space** → **Aortic stenosis**. **Do → Echocardiogram.**
Key 128	**A patient with a classic presentation of MI (sudden onset central chest pain radiating to neck and left shoulder, sweating, vomiting) but the ECG is normal.** → **request troponin.** **If troponin is elevated**

	→ **Myocardial infarction (Non-ST Elevation MI).**
Key 129	◙ **The best initial investigation for chest pain is → ECG.** ◙ **The most appropriate "initial" test for syncopal attacks that are not due to hypoglycemia (no Hx of sweating, tachycardia, hunger, shakiness before the syncope), and not due to epilepsy (No Hx of tongue biting, incontinence during the syncope)** → **ECG** Measuring **lying and standing blood pressure** should also be done to R/O postural hypertension **(Note that** ambulatory blood pressure monitoring is used to diagnose hypertension, not postural hypertension. Thus, not useful in syncopal attacks).
Key 130	◙ The **QRISK3 score** is used to **determine the risk of a cardiovascular event in the next 10 years**. ◙ If QRISK3 is > 10% and the age is < or = 84 → the patient needs to start on **statins** (**atorvastatin**) to reduce the risk of cardiovascular disease. ◙ If QRISK3 is 10%, this means that the patient has 10% chance of developing cardiovascular disease over the next 10 years.

Key 131	◙ Angina, Syncope, Dyspnea, Systolic murmur over the "right" sternal border → **Aortic Stenosis**. ◙ Dyspnea, Orthopnea, Diastolic murmur at the "apex" → **Mitral stenosis**.
Key 132	◙ **SOB**, **Diastolic murmur**, Chest X-ray showing **straightening of the left atrial appendage** Think→ **Mitral stenosis**. (Remember, rheumatic fever is the commonest cause of mitral stenosis. Rheumatic fever is commonly found in developing countries e.g. Syria, Mozambique). ◙ **Complications of MS:** As the left atrium enlarges → **Atrial fibrillation** → **Venous thromboembolism** → **Cerebral infarction**. "imp √". √ Note: left ventricular hypertrophy is a common complication of aortic stenosis.
Key 133	Broad complex tachycardia + Unstable patient (e.g. SBP < 90) → **Synchronized Cardioversion**.

Key 134

Long QT syndrome

Recurrent fainting episodes

+

Prolonged QT intervals on ECG

+

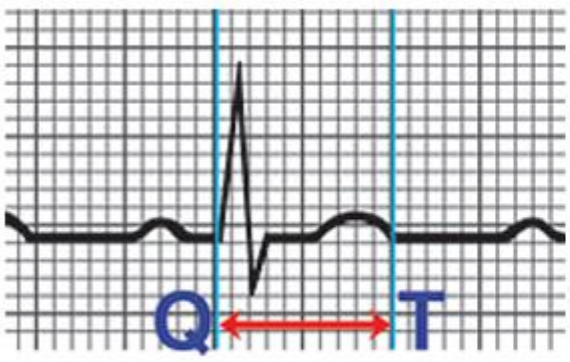
Hx of similar ECG during childhood

• Think → **Congenital long QT syndrome** (Congenital LQTS).

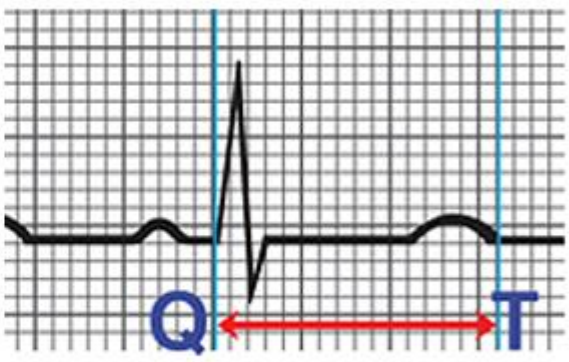
• The most common arrhythmia associated with Congenital LQTS is

→ **Ventricular tachyarrhythmia**.

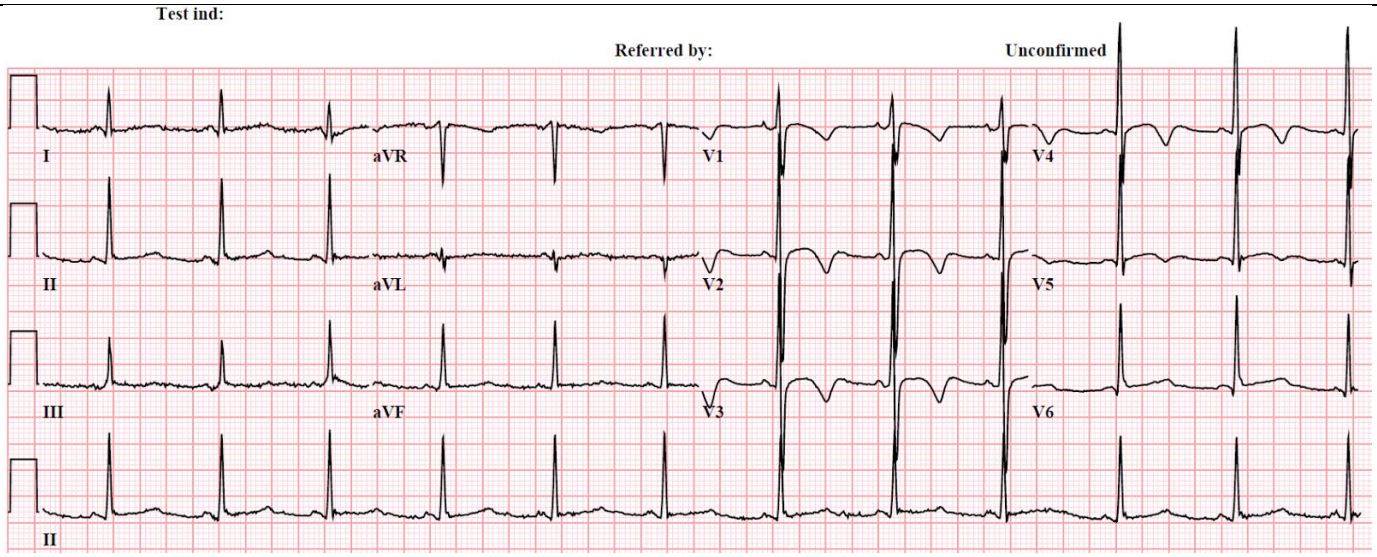
There is risk of ventricular fibrillation. Thus, some patients use long-term beta blocker treatment.



Normal Q-T Interval



Long Q-T Interval

Key 135	 T wave inversion in leads (V1, V2, V3, V4) Indicating → **Myocardial Ischemia**.
Key 136	**A 60 YO man on ramipril for his hypertension has serum potassium of 6.7 mmol/L (Normal: 3.5-5). His ECG shows sinus rhythm without hyperkalemic changes. What is the next step in management?** Although there is no ECG changes of hyperkalemia "tall tented T waves", this patient has **severe hyperkalemia (>6.5)** and the **initial** step is to **protect the heart** and prevent cardiac arrest by giving a medication that antagonizes the cardiac membrane excitability as a temporary measure such as **calcium Gluconate 10%** or **calcium chloride** or **calcium carbonate**.

Key 137	**A 50 YO presents with a swollen left arm. It started suddenly a few hours ago and it is painful. He has fever and breathlessness. O/E, loud S1 and mid-diastolic murmur are heard. There is no left radial pulse felt. Echocardigram is done and it reveals left atrial myxoma. What is the most appropriate management?** → Perform an urgent catheter **Embolectomy**. • A piece of the atrial myxoma has broken off and caused left acute limb ischemia (sudden swollen painful left arm with a loss of radial pulse). • It has been only a few hours and the limb could be saved if embolectomy is performed urgently. • It is perfomed by a interventional radiologist who uses an angiogram in theatre and introduce a catheter to aspirate the emboli "embolectomy". • Note that this is not a blood clot, but a piece of the benign tumor. Thus, thrombolysis and anticoagulation would not be suitable.
Key 138	**Fatigue + Shortness of breath on exertion + Ankle oedema** **Think → Heart failure.**

	Next test → N-terminal (NT)-pro hormone BNP (NT-proBNP). **If raised → Echocardiogram.** --- *• N-terminal (NT)-pro hormone BNP (NT-proBNP) is a non-active prohormone that is released from the same molecule that produces BNP. Both BNP and NT-proBNP are released in response to changes in pressure inside the heart. These changes can be related to heart failure and other cardiac problems.* *• Note: sometimes in heart failure, ECG may be normal.*
Key 139	**A hypertension patient on ramipril. What shoulde be monitored?** **→ Kidney function tests.**
Key 140	**Dizziness + bradycardia + 3rd degree heart block** **◘ Initial management → Atropine.**

◘ **What if he was given atropine but no response?**

Next step would be → Temporary transcutaneous pacemaker.

◘ **Definitive management → Permanent pacemaker.**

• ***Bradycardia*** *is a condition typically defined wherein an individual has a resting heart rate of under 60 beats per minute (BPM) in adults, although some studies use a heart rate of less than 50 BPM.* ***Bradycardia*** *typically does not cause symptoms until the rate drops below 50 BPM.*

• *All symptomatic bradycardia should initially be treated with* ***atropine****.*

If no response → temporary pacemaker (in an emergency setting).

• *Atropine is given 0.5 mg IV push and may be repeated up to a total dose of 3 mg.*

Remember*:*

◙ **1st Degree** Heart Block and **Mobitz type 1** usually

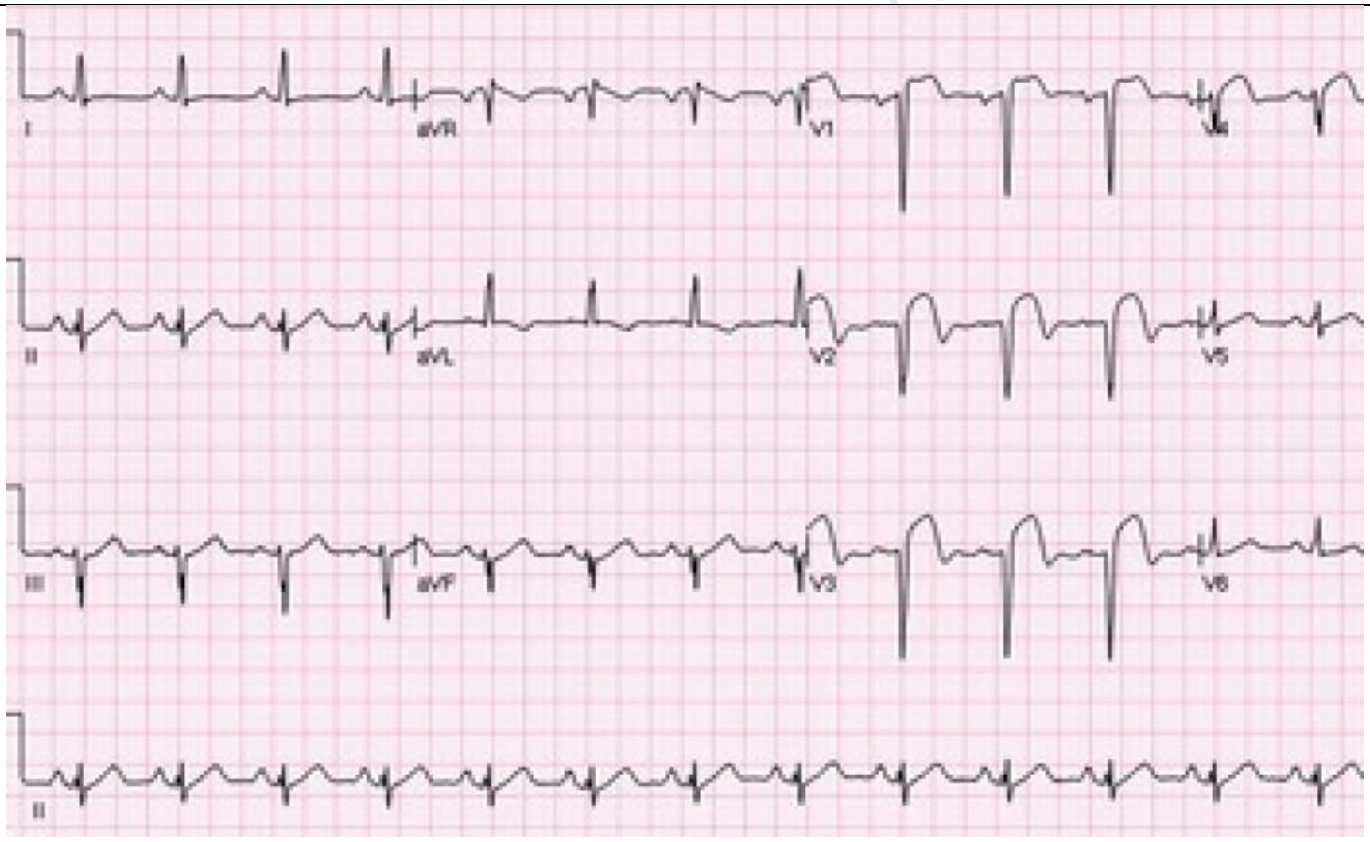
→ **do not require treatment** (as long as the patient is **Asymptomatic**).

◙ **Mobitz type 2** and Complete heart block (**3rd degree** heart block)

→ require **permanent pacemaker.**

• **Initially → Atropine (first choice for all symptomatic bradycardia).**

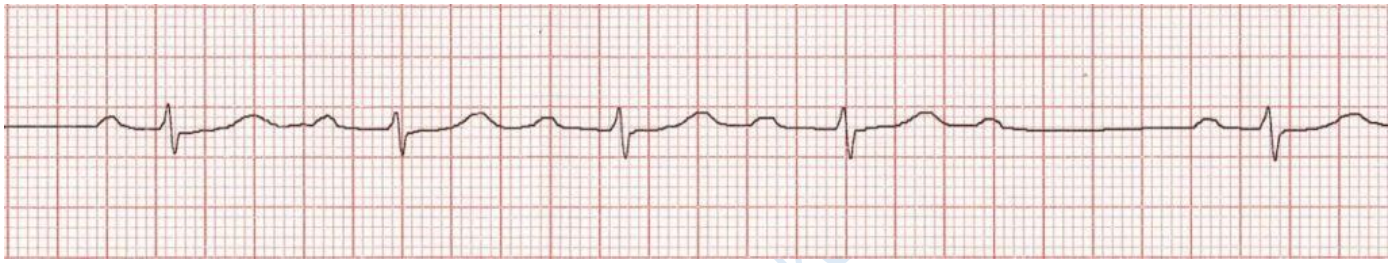
	• **Then → Transcutaneous pacing. (Used to buy time until transvenous pacing is done).** • **Then → Transvenous pacing.** • **Then → Permanent pacing (Pacemaker).**
Key 141	**A 50 YO man presents for advice as his father dies of MI. He smokes 6 cigarettes a day, drinks 14 units of alcohol a week with 2 days free. He eats an estimate of a teaspoon of salt a day. He eats fruits and vegetable. His clinic BP is 138/87.** **What is the best advice to reduce the risk of MI?** **A) Refer to a cardiologist.** **B) Refer to a smoking cessation clinic.** **C) Refer to alcohol cessation services.** **D) Advise to reduce salt.** √ His clinic BP is normal (<140/90). √ Smoking is the major risk factor here. √ His alcohol intake is within the recommended limit. (*no more than 14 units a week with at least 2 days free*).

	√ His salt intake is within the recommended limit. (*Salt intake recommendation for adults in the UK is no more than 6 g per day which is around 1 teaspoon*).
Key 142	If a patient has either systolic BP ≥ 140 and/or diastolic BP ≥ 90 in a **clinic** → **Ambulatory blood pressure monitoring** (**ABPM**) is needed before starting lifestyle modifications and antihypertensive medications.
Key 143	 • **Dx →** **Anterior MI (note ST elevation in V1-V4).**

	• **The likely occluded artery → LAD "left anterior descending artery".**
Key 144	**(Important)** **If the patient of congestive heart failure is already on FUROSEMIDE, Beta blocker, and ACEi and still has lower limb edema → This means that the diuretic is not efficient (the furosemide) → Therefore, one of the following is needed:** • **Increase the dose of the furosemide.** • **Switch the furosemide to either bumetanide or torsemide.** • **Consider admission for IV loop diuretics.**
Key 145	**A 67 YO man with hypertension for 7 years. He is on ramipril "ACEI". However, his BP now is not controlled. Knowing that he had used amlodipine "CCB" in the past but stopped it because of ankle edema, what medication can be added?** • **Choices are (Valsartan, Indapamide, Bisoprolol, Furosemide).** **The right option is → Indapamide (which is a thiazide like diuretic0** **He has started with CCB (step 1) and cannot use it because of ankle edema S/E.** **Then stopped it and went for ACEi ramipril (step 2)** **Now, (step 3) is a thiazide like diuretic e.g., indapamide.** **Note that valsartan is ARBs. It is used instead of ACEi in some cases like those who develop dry cough as a side effect of ACEi.**

Key 146	**√** Arrhythmia causing dizziness and palpitations sometimes but the ECG is normal Do → Ambulatory 24-hour ECG. This is because the arrhythmia occurs in some periods of the day and so the ordinary ECG may be normal. We need ambulatory 24-hour ECG to try to detect the arrhythmia. *Also known as an ambulatory ECG or EKG monitor, a Holter monitor* ***records your heart's activity over 24 to 48 hours or up to 2 weeks while you maintain a diary of your activity to help your doctor identify the cause of your symptoms****.* **√** If Stable angina (chest pain on exercise) is suspected → Exercise ECG "stress test".
Key 147	**A 66 YO diabetic and hypertensive patient developed chest pain radiating to the left arm 1 hour ago. His ECG is normal and his Troponin level is normal (below 12). He was given aspirin. What is the next step?** → **Admit and repeat troponin level after 3 hours of the symptom's onset.** • The patient has arrived to the hospital after **one hour** of the beginning of the chest pain. So, the troponin level was taken only one hour after the chest pain. • **Elevated troponin can be detected within 3 to 4 hours after the onset of myocardial injury**. Serum levels can remain increased for 7 to 10 days for troponin I and 10 to 14 days for troponin T. • Therefore, **a second troponin level is needed after 3 hours of the onset of the chest pain**. Taking into consideration that he is Old + DM + HTN (All RFs for MI).

	◙ **Rules**: **If arrived < 3 hours of the chest pain onset:** **√ troponin is < 12 → repeat after 3 hours of chest pain onset.** **√ troponin is > 30 → correlate with ECG and Hx → treat as ACS.** **If arrived > 3 hours of the chest pain onset:** **√ troponin is < 12 → ACS is unlikely → suspect stable angina and refer to cardiology outpatient or rapid access chest pain clinic.** **√ troponin is > 30 → correlate with ECG and Hx → treat as ACS.**
Key 148	**Pericarditis** **Sharp chest pain + Age 20-55 YO + ECG shows Widespread ST elevation** **+ Recent Hx of upper viral respiratory tract infection** **Think → Acute pericarditis.** **Rx → NSAIDS (eg, Ibuprofen) + Colchicine.** ◙ **Other Criteria of Acute Pericarditis → Central sharp stabbing chest pain relieved by sitting up and leaning forward and worsened by lying down + Pericardial rub.** ◙ **Management of Pericarditis (imp):** √ A full-dose NSAID should be used (eg, aspirin, 2-4 g/d; **ibuprofen** 1200-1800 mg/d; indomethacin 75-150 mg/d); treatment should last at least 7-14 days.

	√ **Colchicine** (as an adjunct to NSAIDs to ↓ inflammation and recurrence).
Key 149	**A 50 YO man is to be started on Modafinil (to counter his extreme sleepiness). A baseline ECG was requested and it shows:**  **What is the most appropriate management?** **A) Atropine** **B) Permanent pacing** **C) Temporary pacing** **D) Reassurance.** • The ECG shows PR prolongations followed by a dropped beat. • This is a second-degree heart block (Mobitz type 1). Also called (Wenckebach). • Asymptomatic patients with this type of heart block do not need specific Rx.

Key 150

A 58-year-old man had clinic blood pressure of 162/95 mmHg. He was asked to record blood pressure at home (ABPM). He returns to the clinic next week with an average ambulatory blood pressure reading of 155/92 mmHg. He does not have a significant medical history. What is the most appropriate action?

A) Repeat ABPM for another week.
B) Prescribe enalapril.
C) Prescribe felodipine.
D) Prescribe indapamide.
E) No treatment is required, only encourage lifestyle modifications.

• Since his ambulatory BP is ≥ 150/95 (His SBP is the high portion in this case)

→ he needs treatment. (Confirmed stage 2 HTN).

(f ABPM or HBPM ≥ 150/95 mmHg (i.e., confirmed stage 2 or higher hypertension) → Always treat.)

• Since he is ≥ 55 YO → the first step is Calcium channel blocker (CCB).

→ Felodipine (Felodipine is a CCB).

Important notes regarding [When to treat Hypertension?]:

Stage	Criteria
Stage 1 HTN	**Clinic BP** ≥ 140/90 and subsequent **ABPM** daytime average **or HBPM** average BP ≥ 135/85
Stage 2 HTN	**Clinic BP** ≥ 160/100 and

	subsequent **ABPM** daytime or **HBPM** average BP ≥ 150/95
Stage 3 HTN	**Clinic** systolic BP ≥ 180 mmHg, **or clinic** diastolic BP ≥ 110 mmHg

◙ If a patient is completely free and has a stage 1 Hypertension

→ Lifestyle and Diet Modification + review (Follow up).

◙ In a patient with stage 2 hypertension at a clinic (Clinic BP ≥ 160/100)

→ Before commencing antihypertensive drugs, request either ABPM or HBPM.

◙ If ABPM or HBPM ≥ 150/95 mmHg (i.e., confirmed stage 2 or higher hypertension) → Always treat.

◙ For patients < 40 years and with stage 2 hypertension or higher

→ Consider a specialist referral to exclude secondary causes of HTN.

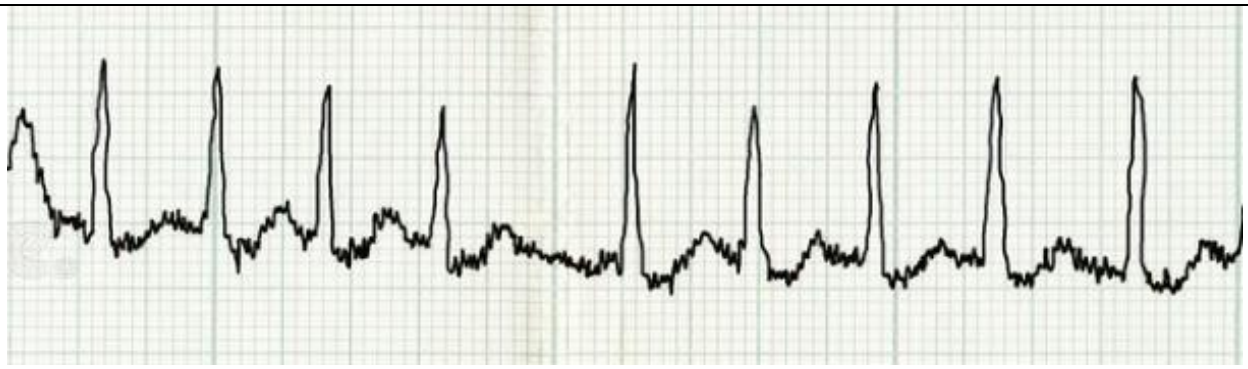
Remember, the first step in managing HTN is as follows:

- **White + < 55 YO** → start with **ACEi**/**ARBs** as a step 1 management of HTN.
- **White + > 55 YO** → start with **CCB** as a step 1 management of HTN.
- **Afro-Caribbean + any age** → start with **CCB** as a step 1 management of HTN.

- Example of **ACEi** → Enalapril.

	- Example of **ARBs** → Valsartan. - Example of **CCB** → Amlodipine, Felodipine. - Examples of **thiazide diuretics** → chlorthalidone (12.5-25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily) - ~~Bendroflumethiazide~~ is a thiazide like diuretic; however, it is no longer recommended by NICE as an antihypertensive.
Key 151	**Patients with aortic stenosis (systolic murmur at the right second ICS) are liable to fainting episodes that are called → Syncope.** **Other features → Dyspnea, Angina.**
Key 152	**After Myocardial Infarction, Mitral regurgitation may occur as a complication (**pansystolic murmur at the apex +Bi-basal crackles + Shortness of breath**).** **The most appropriate investigation to determine this mitral regurg is** **→ Echocardiogram.**
Key 153	**In diabetic patients who have heart failure "HF" with reduced ejection volume** **Give → SGLT2 inhibitors such as Dapagliflozin, Empagliflozin** (When SGLT2 inhibitors are added to the medications of HF which are B-blockers, ACE inhibitors, Aldosterone antagonist → they reduce cardiovascular death). So, in **DM** with **heart failure**, use metformin and **flozin** family.

Key 154	A 72-year-old man presents to a GP clinic to complain of repetitive dizziness and palpitations over the past few weeks. They mostly occur while walking long distances and they resolve after a few minutes. He has a background history of diabetes mellitus types 2 and hypertension. He is vitally stable. His current ECG shows normal sinus rhythm. He denies any chest pain, fainting or shortness of breath. What is the most appropriate investigation? A) Echocardiogram. **B) Ambulatory 24-hour ECG (Holter monitor).** C) Exercise ECG (Stress test). D) Random blood glucose. E) Blood pressure monitoring. • **Exercise ECG (stress test)** is preferred for stable angina. Here, no chest pain! • **Echo**: could be requested if the current ECG is abnormal. Here, it is normal. • Also, if you think this might be **aortic stenosis (AS)** that requires Echo to be diagnosed, then you are mistaken. **AS** presents with ejection systolic murmur over the right second intercostal space + Fainting episodes (syncope) ± Dyspnea ± Angina (chest pain). None of these features are present! • This patient has **dizziness** and **PALPITATIONS** mostly during walking, which may indicate **hidden arrhythmia** which develops while walking. • The arrhythmia can be detected by ECG. However, the current ECG is normal. Thus, **ambulatory 24-hour (Holter) ECG** is needed.
Key 155	**A 60-year-old has just finished his appendectomy surgery and developed dizziness, palpitations, tachycardia and hypotension (BP: 80/50). His ECG:**



What is the most appropriate diagnosis and management?

- The ECG + (palpitations, tachycardia. Dizziness) suggest **atrial fibrillation**.
- Since the **AF** has just started and the patient is **hypotensive** (ie, haemodynamically **unstable**) → **Cardioversion**. √
- If cardioversion is not in the option → **IV Amiodarone**. √
- Another possible answer is IV Flecainide (**IV**, not Oral).

(Careful: **beta-blockers** are first line in **STABLE** patients with AF)!

Key 156

- Remember that the **initial treatment for symptomatic bradycardia** (lightheadness, syncope -fainting-, dizziness) is → IV atropine.

(Given 0.5 mg IV push and may be repeated every 3-5 minutes up to a total dose of 3 mg).

	• Rx is required if the patient is symptomatic even if the ECG shows first-degree heart block. **As long as he has symptoms** and **bradycardic**, start with atropine. • After initial stabilisation of the patient by atropine, he should then be prepared for temporary pacemaker and assessed for whether or not he needs a long-term permanent pacemaker.
Key 157	**Important Points to Remember on Ischemic Stroke** • The antihypertensive of choice in patients with diabetes mellitus is → ACE inhibitors (eg, ramipril). **• In patients with a history of ischemic stroke, the long-term management to prevent further stroke (2ry prevention of ischemic stroke) is as follows:** **√ Control Blood Pressure**. Remember, if he has diabetes, pick or add ACEi eg, ramipril. **√ Statins** (for All patients regardless of their cholesterol baseline level). **√ Ani-platelets** (or) **Anti-coagulation**: (Based on presence or absence of AF):

· If there is Atrial Fibrillation → Anticoagulants: Warfarin [or] DOAC (Dabigatran/ Apixaban/ Rivaroxaban/ Edoxaban). DOAC is now preferred.

(Warfarin is almost obsolete nowadays. DOAC is recommended instead).

· If No Atrial Fibrillation → Antiplatelets: Clopidogrel 75 mg OD.

Important: What if a patient with a history of stroke is already on warfarin and presents with a new onset atrial fibrillation?

Since he is already on an anticoagulant, he should continue on warfarin and should not be switched to DOAC (eg, Edoxaban) unless his INR is abnormal.

Scenario (1) that tests the above notes (important).

A 65 YO man presents for medication review. He has a history of ischemic stroke a few months ago. His medical background includes atrial fibrillation, hypertension and type 2 diabetes mellitus. His ECG still shows arterial fibrillation. He is on warfarin, lercanidipine (CCB), atorvastatin, and metformin. His current blood pressure is 160/100. What is the most appropriate action?

A) Switch warfarin to Edoxaban (DOAC).

B) Continue warfarin and add clopidogrel.

C) Stop lercanidipine and start ramipril.

D) Continue lercanidipine and add ramipril.

E) Continue lercanidipine and add bendroflumethiazide.

The right answer is (D): continue lercanidipine (CCB) and add ramipril (ACEi).

• Since he is **diabetic**, **ACEi** is of choice to control HTN.

• Since he is already on an antihypertensive (which is lercanidipine; a calcium channel blocker) and **his hypertension is still uncontrolled**, go to step 2 and add a second antihypertensive (**ACE inhibitor** eg, **ramipril**).

• We **do not shift** from warfarin to DOAC unless his INR is not normal. Here, no mention of INR. Thus, continue on warfarin (option A is wrong).

• Since he has AF, an anticoagulant (eg, warfarin, DOAC) is given; Not an antiplatelet (eg, clopidogrel).

Scenario (2) that tests the above notes (important).

A 61 YO man presents for medication review. He has a history of ischemic stroke a few months ago. His medical background includes hypertension and type 2 diabetes mellitus. His ECG now shows arterial fibrillation. He is on clopidogrel, amlodipine, atorvastatin, and metformin. What is the most appropriate action?

A) Switch amlodipine to ramipril.

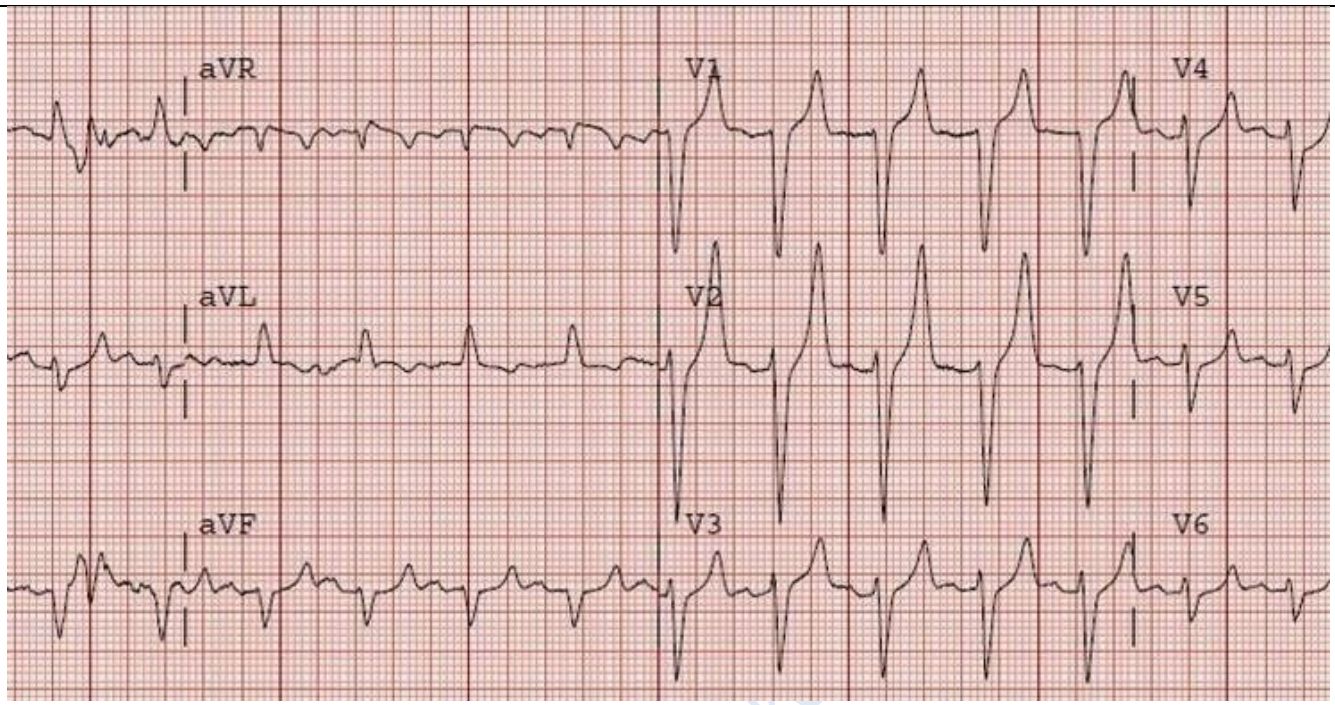
B) Continue clopidogrel and add apixaban.

C) Stop clopidogrel and start edoxaban.

D) Continue amlodipine and add ramipril.

E) Continue amlodipine and add bendroflumethiazide.

	The right answer is ©: Stop clopidogrel and start a DOAC eg, edoxaban. • He did not have atrial fibrillation before, that's why he was on clopidogrel (and not warfarin or DOAC). • Since he is now having atrial fibrillation, switch clopidogrel to an anticoagulant such as warfarin or DOAC (eg, edoxaban, apixaban). DOAC is preferred over warfarin. • His blood pressure is not mentioned so we assume it is controlled and therefore no need to add an additional antihypertensive (even if he is diabetic, he is already on amlodipine and controlled).
Key 158	• In **rhabdomyolysis**, acute kidney injury and hyperkalemia can develop. • A patient might be found on the floor after a fall for several hours. • A patient can be found stuck under a heavy object for several hours. • Hyperkalemia can develop and **ECG** may show **tall tented T waves**.



Tall Tented T waves → Hyperkalemia

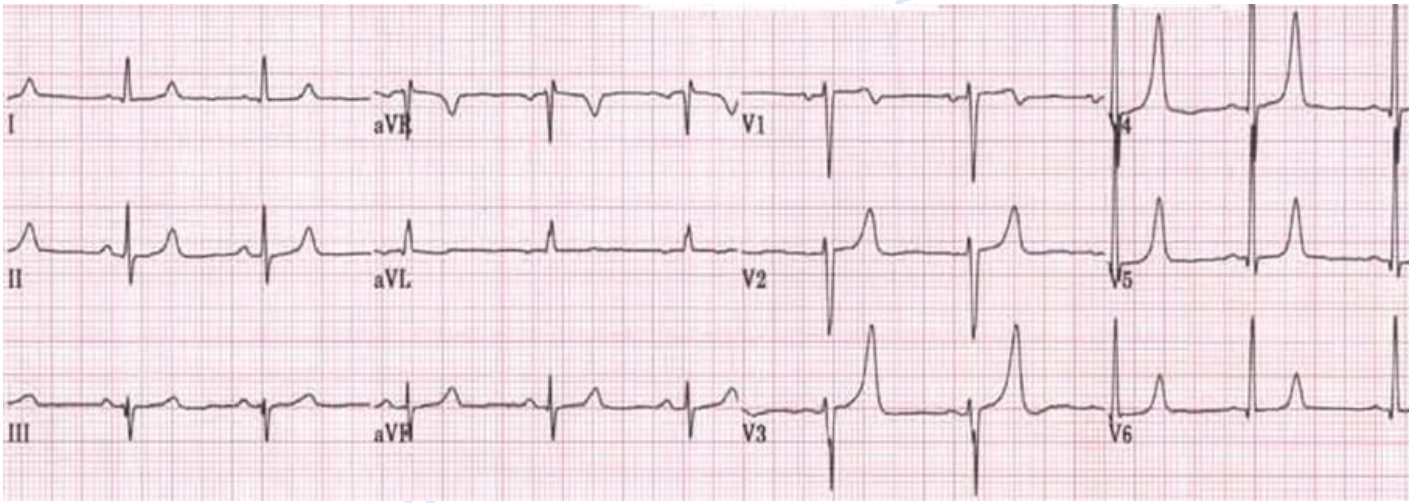
• **Important**: the investigation that would likely be **abnormal** in this case is

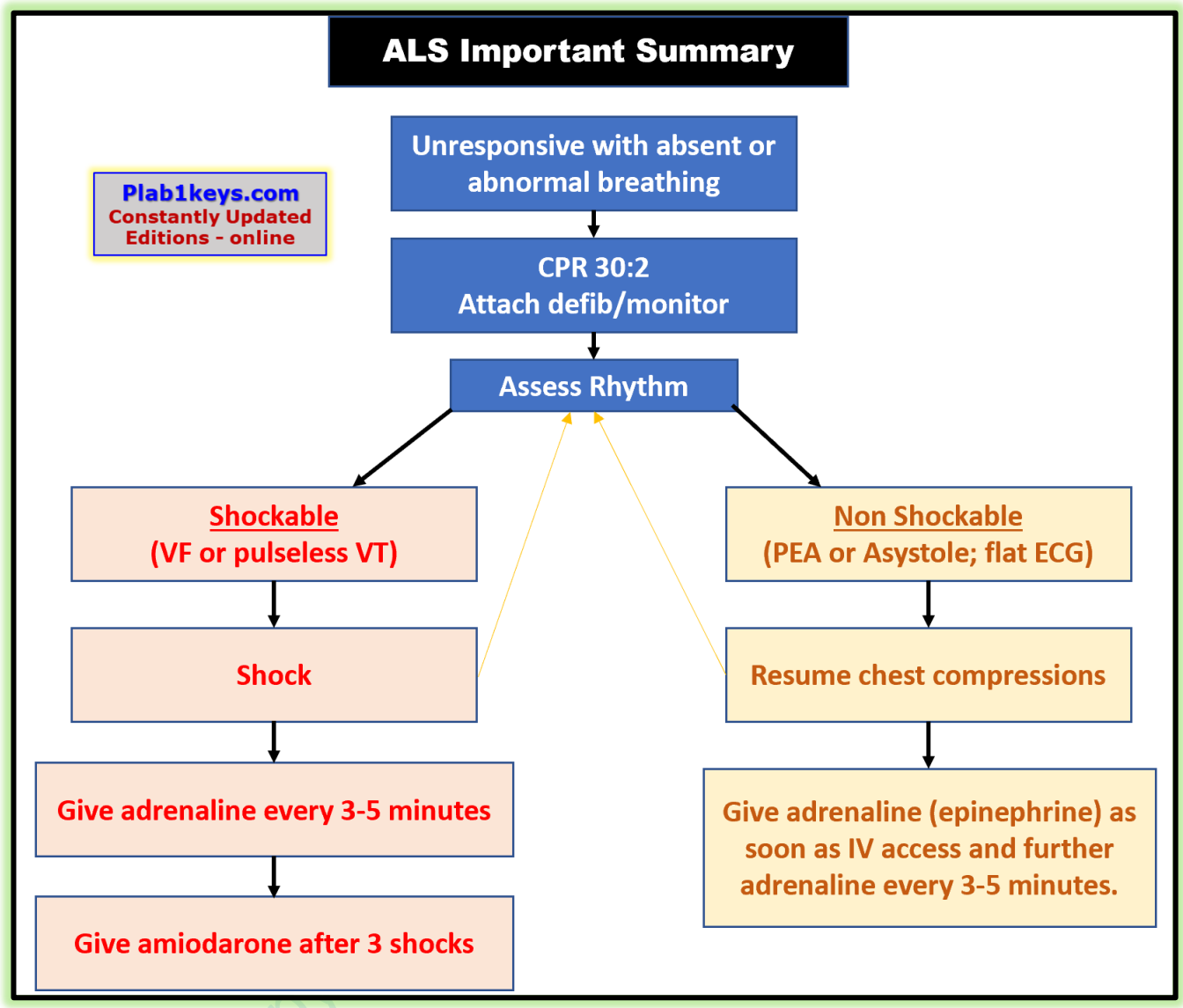
→ Urea and electrolytes.

Urea and electrolyte panel includes:

→ Urea, creatinine, sodium, potassium, eGFR.

Key 159	• **Consuming alcohol** (and also **tobacco**) excessively can lead to transient **palpitations** several times a day (holiday heart syndrome). • The ECG in this case would be unremarkable.

	• The best action is to → Advise on lifestyle modifications (decreasing alcohol and smoking would resolve palpitations completely).
Key 160	• A patient with **DM type 1** who presents with abdominal pain, vomiting, lethargy, increased thirst and urination, and fruity smelling breaths is likely suffering from a complication called **diabetic ketoacidosis** (**DKA**). • One of the manifestations that would be seen in DKA is: → **Hyperkalemia** (ECG might show **tall tented t waves**)  Tall Tented T waves (Hyperkalemia).
Key 161	**ALS important points**



◙ Example (1):

A 50-year-old man was found unconscious with a absent pulse and undetectable blood pressure. His ECG reading is flat. What is the best management?

	→ Start CPR (Chest Compressions). Important: If CPR is not among the options, what's to pick? → Give adrenaline (Epinephrine) 1 mg IV. (A flat ECG reading means asystole which is a **non**-shockable rhythm). --- ◙ **Example (2):** A 50-year-old man was found unconscious with a absent pulse and undetectable blood pressure. His ECG reading shows VT. What is the best management? → Deliver a shock. (VT in a pulseless patient and VF requires immediate shock delivery).
Key 162	**A few days after myocardial infarction (especially inferior MI):** • **Pan-systolic** murmur over the apex Think → Mitral regurgitation.

	• **Pan-systolic** murmur over the lower left sternal border Think → Tricuspid regurgitation Or ventricular septal rupture. (This is due to a possible dysfunction of papillary muscles of the valve). **Q) Since these 2 conditions have the same type of murmur, how to differentiate?** **√** Ventricular septal defect occurs acutely (within a few days) after MI, and it also presents with hypotension (shock). **√** Tricuspid regurgitation tends to develop over weeks to months after MI, and hypotension is not a feature. - In both conditions, do → **Echocardiogram**.
Key 163	**Important notes on the management of Atrial Fibrillation:** (Atrial fibrillation = narrow-complex irregularly irregular tachycardia). • First line (if hemodynamic **stable**) → **Beta-blockers** (eg, **metoprolol**). • If hemodynamic **unstable** (eg, hypotension, chest pain, altered mentation) → Start with **electrical cardioversion**. If still not improved → **IV amiodarone**.

Key 164

An Important Question on Hypertension Management (Reminder):

A 67-year-old man presented to the GP surgery to check on his blood pressure. It is found to be 155/95 mmHg. He was asked to measure his blood pressure at home several times. On the next appointment, the average of his home blood pressure measurements is 146/93 mmHg. His 10-year risk of developing cardiovascular disease is calculated and is found to be 6%. He does not smoke and otherwise healthy. What is the most appropriate management?

A) Start ACE inhibitors.
B) Start calcium channel blockers.
C) Offer lifestyle advice and modification.
D) Start statin.
E) Reassure and no intervention is required.

Answer → C.

Since his home blood pressure is ≥ 140/90, but < 150/95 mmHg.
→ He has **stage (1) hypertension**.

Stage	Criteria
Stage 1 hypertension	**Clinic BP** ≥ 140/90 mmHg and subsequent **ABPM** daytime average **or** **HBPM** average BP ≥ 135/85 mmHg
Stage 2 hypertension	**Clinic BP** ≥ 160/100 mmHg and subsequent **ABPM** daytime or **HBPM** average BP ≥ 150/95 mmHg

	Stage 3 "Severe hypertension"	**Clinic** systolic BP ≥ 180 mmHg, **or clinic** diastolic BP ≥ 110 mmHg

♠ When to Treat Stage 1 Hypertension?

• Treat if the patient's age is < 80 years **AND** + any of the following:

Target organ damage, established cardiovascular disease, renal disease, diabetes (DM) or a 10-year **cardiovascular risk equivalent to (QRISK2) ≥ 10%.**

◙ **Note: If a patient is completely free and has a stage 1 Hypertension**

→ **Lifestyle and Diet Modification + review (Follow up).**

◙ **Example: If a patient a stage 1 Hypertension and his QRISK2 ≥ 10?**

→ **Discuss initiating antihypertensive therapy.**

Key 165	A 53-year-old man from South Asia presents with headache and blurry vision for sometimes. His blood pressure is found to be 149/ 101 mmHg. He was asked to have blood pressure measurements at home. His average blood pressure of 14 home readings is 153/97 mmHg. What is the most appropriate management? A) lifestyle modification only.

B) No management required.

C) Ramipril.

D) Indapamide.

E) Amlodipine.

Answer → C.

- He has a stage 2 hypertension.
- Since he is < 55 YO → Start with ACE inhibitor (eg, ramipril).

Stage	Criteria
Stage 1 hypertension	**Clinic BP** ≥ 140/90 mmHg and subsequent **ABPM** daytime average **or** **HBPM** average BP ≥ 135/85 mmHg
Stage 2 hypertension	**Clinic BP** ≥ 160/100 mmHg and subsequent **ABPM** daytime or **HBPM** average BP ≥ 150/95 mmHg
Stage 3 "Severe hypertension"	**Clinic** systolic BP ≥ 180 mmHg, **or** **clinic** diastolic BP ≥ 110 mmHg

- If ABPM (Ambulatory BP monitoring) or HBPM (Home BP monitoring) is ≥ 150/95 mmHg (i.e., **stage 2** or **higher** hypertension) → **Always treat**.

▢ Step 1 in Hypertension Management:

√ Patients < 55-years-old → start with ACE inhibitor (**A**) or ARBs.

√ Patients ≥ 55-years-old **or** of Afro-Caribbean origin "**of any age**"

→ start with Calcium channel blocker.

In other words:

- **White + < 55 YO** → start with **ACEi**/**ARBs** as a step 1 management of HTN.
- **White + > 55 YO** → start with **CCB** as a step 1 management of HTN.
- **Afro-Caribbean + any age** → start with **CCB** as a step 1 management of HTN.

▢ Step 2 → Both: **A**CE inhibitor + **C**alcium channel blocker (**A** + **C**).

▢ Step 3 → Add a Thiazide **D**iuretic (**D**).

- Example of **ACEi** → Enalapril, Ramipril.
- Example of **ARBs** → Valsartan, Losartan, Candesartan.
- Example of **CCB** → Amlodipine, Felodipine.
- Examples of **thiazide diuretics** → chlorthalidone (12.5-25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily).

Answer: This patient has stage **2** hypertension, and he is **< 55** years old, so → start with an ACE inhibitor (eg, **ramipril, enalapril**).

Key 166

Remember (Regarding Hypokalemia):

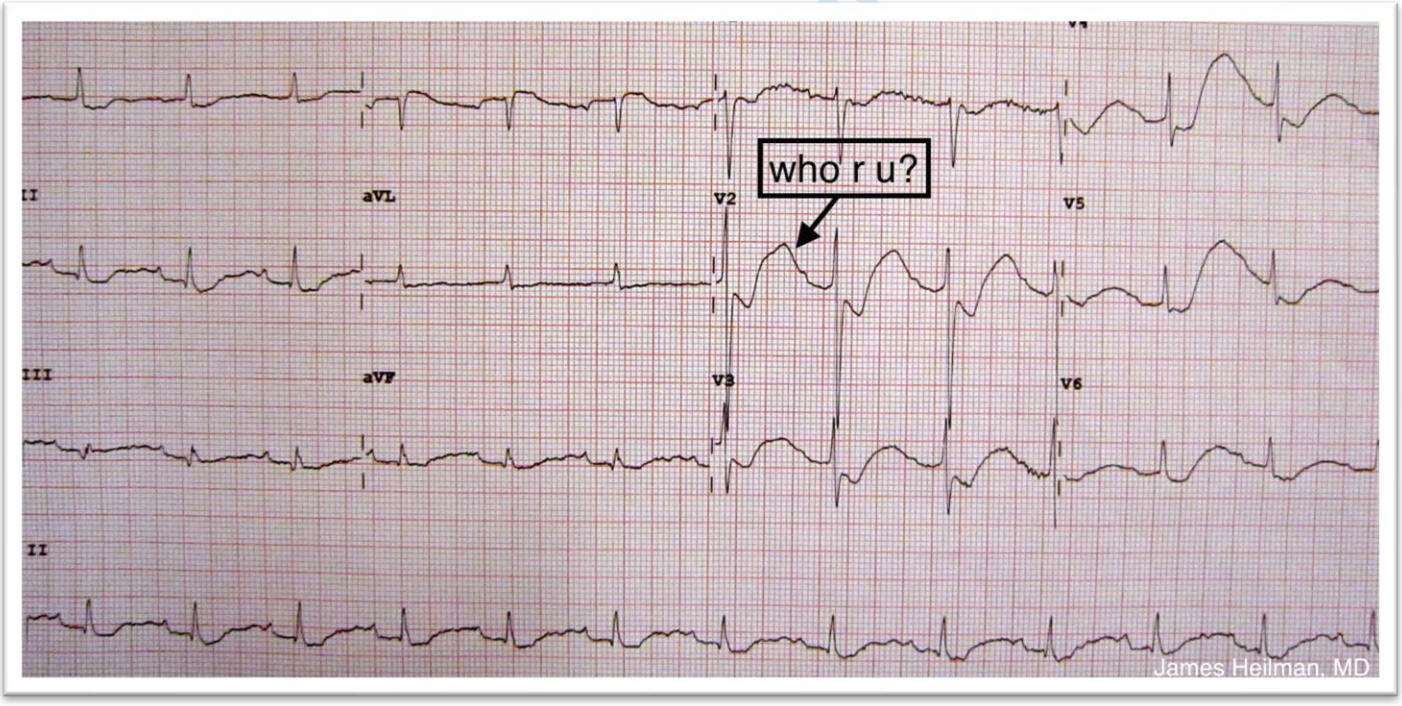
√ Severe vomiting can cause hypokalemia.

√ Hypokalemia features include muscle weakness and cramping.

√ ECG features of hypokalemia involve:

U wave (an additional wave after the "T" wave).

Others: Inverted T wave, Depressed ST segment.



Management of hypokalemia

1) Oral or IV **Potassium chloride** (based on severity), e.g. if K+ <2.5 → IV.
2) Stop/ Treat the cause (e.g. stop furosemide, thiazide like diuretics).

Key 167

Important Notes on Hyperkalemia

A patient presents with both (Hyperkalemia; eg, potassium 6.9 mmol/L) associated with broad QT complexes on ECG

AND

Bradycardia (HR is < 60 bpm).

What to treat first?

→ Treat **hyperkalemia** **first (**by giving **IV** **calcium Gluconate 10%** or **calcium chloride** or **calcium carbonate**).

As initial intervention, treating hyperkalemia with IV calcium -which would stabilize the cardiac membrane and prevent cardiac arrest- is more critical than treating bradycardia with atropine.

Notes:

- If K^+ is **≥ 6.5** mmol/L (ie, severe) → treat with IV calcium (even if ECG is normal).
- If K^+ is **6 – 6.4** (ie, moderate) → treat with IV calcium (if there is ECG changes).

Key 168

Important Note on Primary Prevention of Cardiovascular Events

For all people **≤ 84** YO who have **QRISK3** Score of **≥ 10**

Offer → Statins (eg, atorvastatin) (for 1ry prevention of CVD).

Key 169	**Systolic murmur at the left lower sternal border worsened by Valsalva manoeuvre + ECG evidence of left ventricular hypertrophy (LVH)** Think → Hypertrophic cardiomyopathy (HOCM).
Key 170	**A few days after MI:** • Pansystolic murmur best heard at the left lower sternal border. • Shock (hypotension and tachycardia). • Heart failure/ pulmonary edema (eg, bibasilar crackles). Think → **Ventricular septal defect/ rupture**. **Why NOT tricuspid regurgitation (TR)?** √ Tricuspid regurgitation can occur as a complication of MI, but it develops over weeks to months, not acutely and within a few days after MI. √ Also, hypotension and shock are another sign of ventricular septal defect, not TR.
Key 171	**Quick Notes to Remember:** ◙ If QRISK3 is ≥ 10% → Start atorvastatin "regardless of lipid profile level". ◙ High doses of **Citalopram**, (a SSRI), can cause prolonged QT intervals, which can lead to **Torsade De Pointes**.

Key 172

Ventricular Septal Rupture (VSR) Post-Myocardial Infarction

Ventricular septal rupture (VSR) is a rare but serious mechanical complication that occurs following a myocardial infarction (MI). It typically develops 3–5 days after the MI due to necrosis of the interventricular septum, which leads to a communication between the left and right ventricles.

Key Features:

√ New Murmur: A characteristic **pan-systolic murmur**, best heard at the left lower sternal border (LLSB).

√ Hemodynamic Instability: VSR often leads to cardiogenic shock, evidenced by hypotension (e.g., blood pressure 90/60 mmHg), diaphoresis, and jugular venous distension.

√ Respiratory Symptoms: Pulmonary crackles on auscultation, indicating pulmonary edema due to the acute hemodynamic changes.

Diagnosis:

→ **Echocardiography**: This is the definitive diagnostic tool. It confirms the presence and severity of the septal rupture and helps in planning further management.

Clinical Context:

• VSR should be suspected in any patient who develops a new pan-systolic murmur a few days after an MI, especially if accompanied by signs of hemodynamic instability.

• The murmur is typically loudest at the left sternal border.

• Immediate intervention is crucial as the condition rapidly progresses, often requiring surgical repair.

Differential Diagnoses:

Acute Pericarditis: Usually presents with pleuritic chest pain and a pericardial rub, not a murmur.

	Dressler Syndrome: A form of post-MI pericarditis that occurs weeks after the event, presenting with fever and pleuritic chest pain. **Left Ventricular Aneurysm**: Occurs weeks to months post-MI, presenting with heart failure symptoms rather than a new murmur. **Mitral Regurgitation**: While it may present with a pan-systolic murmur, the murmur typically radiates to the axilla. In summary, VSR post-MI is a critical diagnosis characterized by a pan-systolic murmur at the left sternal border and hemodynamic instability. Early diagnosis and intervention are essential to prevent fatal outcomes.
Key 173	**Long QT Syndrome and Ventricular Tachyarrhythmia** **Overview**: A 19-year-old male presents with recurrent episodes of syncope, particularly during exercise. His ECG reveals a prolonged QT interval, suggesting **long QT syndrome (LQTS)**, which is associated with a high risk of ventricular tachyarrhythmias, such as **torsades de pointes**. This condition often presents in childhood and is triggered by physical activity, which can precipitate dangerous arrhythmias, leading to syncope or even sudden cardiac death.

Key Features:

- **Prolonged QT Interval**: Identified on ECG, which increases the risk of life-threatening arrhythmias.
- **Syncope with Exercise**: Episodes are commonly triggered by physical exertion in patients with LQTS.
- **Ventricular Tachyarrhythmias**: The most likely arrhythmia in this case, associated with the prolonged QT interval and syncope.

Differential Diagnosis:

- **Sick Sinus Syndrome**: Causes bradyarrhythmias and syncope due to slow heart rates, but not associated with prolonged QT intervals.
- **Ventricular Ectopics**: Extra heartbeats that are common in young individuals and often benign. These are not linked to syncope or prolonged QT interval unless very frequent.

In summary, this patient's presentation is highly suggestive of **long QT syndrome**, and the likely arrhythmia responsible for his symptoms is **ventricular tachyarrhythmia** such as **Torsades de Pointes**. Early identification and management are crucial to prevent severe complications, including sudden cardiac death.

Important Note:

Torsades de pointes is a type of **ventricular tachyarrhythmia** that is associated with a prolonged QT interval. It is the hallmark and most dangerous complication of congenital long QT syndrome (LQTS).

Key 174

Simplified Hypertension Management Summary

Classification of Hypertension:

1. **Stage 1 Hypertension**:
   - Clinic BP: ≥ 140/90 mmHg.
   - Confirmed by Ambulatory BP Monitoring (ABPM) or Home BP Monitoring (HBPM): Daytime average BP ≥ 135/85 mmHg.
2. **Stage 2 Hypertension**:
   - Clinic BP: ≥ 160/100 mmHg.
   - Confirmed by ABPM or HBPM: Daytime average BP ≥ 150/95 mmHg.
3. **Stage 3 (Severe) Hypertension**:
   - Clinic systolic BP: ≥ 180 mmHg **OR**
   - Clinic diastolic BP: ≥ 120 mmHg.

Stage	Clinic BP	ABPM/HBPM Average
Stage 1 Hypertension	≥ 140/90 mmHg	Daytime average BP ≥ 135/85 mmHg
Stage 2 Hypertension	≥ 160/100 mmHg	Daytime average BP ≥ 150/95 mmHg
Stage 3 (Severe) Hypertension	Systolic BP ≥ 180 mmHg OR Diastolic BP ≥ 120 mmHg	N/A

Management of Hypertension:

1. **For Stage 1 Hypertension**:

√ Treat if **<80 years** of age and one or more of the following conditions:

- Target organ damage.
- Established cardiovascular disease.
- Renal disease.
- Diabetes.
- 10-year cardiovascular risk of ≥10%.

√ For patients **>80 years** of age, treatment decisions are individualized, considering overall health, frailty, and potential risks of treatment.

√ If the patient is "completely healthy" and has no significant comorbidities, **lifestyle modifications** (such as salt reduction, regular exercise, and smoking cessation) are recommended. **Antihypertensive treatment** is generally **not required** unless additional risk factors (e.g., organ damage or high cardiovascular risk) are present.

2. **For Stage 2 Hypertension**:
   - **Patients <55 years**: Treat with an ACE inhibitor.
   - **Patients ≥55 years** or of **Afro-Caribbean origin**: Start with a calcium channel blocker.
   - If the patient's blood pressure remains above 140/90 mmHg on treatment, escalate to step 2.

Management of Stage 1 and Stage 2 Hypertension

Condition/Patient Type	Treatment Approach
Stage 1 Hypertension	
Age < 80 years with risk factors	Treat with antihypertensives if one or more of the following: - Target organ damage. - Cardiovascular disease. - Renal disease. - Diabetes. - 10-year cardiovascular risk ≥ 10%.
Age > 80 years	Individualized treatment based on overall health, frailty, and potential risks.
No comorbidities ("completely healthy")	Advice on lifestyle modifications (e.g., diet, exercise, no smoking); no antihypertensive treatment unless high risk
Stage 2 Hypertension	
Patients < 55 years	Start with an ACE inhibitor

Patients ≥ 55 years **or** Afro-Caribbean origin	Start with a calcium channel blocker.
Escalation	If BP remains >140/90 mmHg after initial treatment, move to step 2 treatment as in the following table.

Stepwise Drug Treatment for Essential Hypertension in Non-Diabetic Patients:

Step	Patient Characteristics	Treatment Options
Step 1	Age <55	Start with ACE inhibitor.
	Age ≥55 or Afro-Caribbean origin	Start with Calcium Channel Blocker (CCB).
Step 2	BP uncontrolled in Step 1	Add a second drug: ACE inhibitor, CCB, or Thiazide-like diuretic.
Step 3	BP uncontrolled in Step 2	Combine all three: ACE inhibitor + CCB + Thiazide-like diuretic.

Key Notes:

- **Patients with diabetes**: Start with **ACE inhibitors or ARBs**, regardless of age or ethnicity, to protect kidney function.
- **Lifestyle Modifications**: Encourage lifestyle changes in all patients, including:
  - Salt reduction.
  - Regular exercise.
  - Stress management.
  - Limiting alcohol intake.
  - Smoking cessation.

Common Antihypertensive Examples:

- **ACE Inhibitors**: Ramipril, Lisinopril.
- **Angiotensin II Receptor Blockers** (**ARBs**): Losartan, Candesartan.
- **Calcium Channel Blockers** (**CCBs**): Amlodipine, Diltiazem.
- **Thiazide-like Diuretics**: Indapamide, Chlorthalidone.

Extra Question – Resistant Hypertension:

What is the next step in managing resistant hypertension according to NICE UK guidelines if blood pressure remains uncontrolled after trying an ACE inhibitor/ARB, a calcium channel blocker, and a thiazide-like diuretic (ie, **after trying all steps 1, 2, and 3 but hypertension remains uncontrolled**)?

Answer:

According to **NICE UK guidelines (NG136)**, if blood pressure remains uncontrolled after steps 1, 2, and 3, the next step in managing resistant hypertension (Step 4) involves the following:

1. **Add Low-Dose Spironolactone**:
   - If the patient's **potassium levels are ≤ 4.5 mmol/L**, add **spironolactone** at a dose of 25 mg once daily.
2. **Alternative Option**:
   - If the patient's **potassium levels are > 4.5 mmol/L** or if **spironolactone** is not tolerated, consider using an **alpha-blocker** (e.g., **Doxazosin**) or a **beta-blocker** (e.g., **Atenolol**).
3. **Referral to Specialist**:
   - If the blood pressure remains uncontrolled despite these interventions, refer the patient to a **hypertension specialist** for further evaluation and management.

4. **Monitoring**:
   - Regular monitoring of **potassium levels** and **renal function** is necessary, especially when using spironolactone.

This approach is based on the NICE NG136 guidelines for managing **resistant hypertension** in adults.

Hypertension Case Study Scenarios:

Scenario 1:

Patient: A 65-year-old man presents with a clinic blood pressure of 155/95 mmHg. He has a history of type 2 diabetes and is of Afro-Caribbean origin.

Answer: Start with an **ACE inhibitor** (e.g., **Ramipril**, **Lisinopril**) or an **ARB** (e.g., **Losartan**, **Candesartan**).

Reasoning: For diabetic patients, ACE inhibitors or ARBs are the first-line treatment regardless of age or ethnicity due to their kidney-protective effects. The presence of diabetes takes precedence over ethnicity in this case.

Scenario 2:

Patient: A 45-year-old woman presents with a clinic blood pressure of 150/95 mmHg. She has no history of organ damage, cardiovascular disease, renal disease, or diabetes. She is otherwise healthy and does not smoke.

Answer: Recommend **lifestyle modifications** such as reducing salt intake, increasing exercise, and quitting smoking.

Reasoning: In a healthy patient without risk factors or comorbidities, antihypertensive medication is not required for **Stage 1 hypertension** (clinic BP ≥ 140/90 mmHg but <160/100 mmHg). Lifestyle changes are the first recommendation unless there are additional risk factors like organ damage, diabetes, or cardiovascular disease.

Scenario 3:

Patient: A 50-year-old woman has been on **Ramipril** (ACE inhibitor) for 6 months for her **Stage 2 hypertension** (clinic BP ≥ 160/100 mmHg), but her blood pressure remains elevated at 160/95 mmHg. Her doctor added **Amlodipine**

(calcium channel blocker), but her blood pressure is still above 140/90 mmHg after another 3 months.

Answer: Add a **thiazide-like diuretic** (e.g., **Indapamide**, **Chlorthalidone**).

Reasoning: After trying an ACE inhibitor and a calcium channel blocker (Step 1 and Step 2), adding a third drug, such as a thiazide-like diuretic, is the next step to achieve better blood pressure control.

Scenario 4:

Patient: A 40-year-old man presents with a clinic blood pressure of 170/100 mmHg. He has no significant medical history, no diabetes, and no organ damage.

Answer: Start with an **ACE inhibitor** (e.g., **Lisinopril**, **Enalapril**).

Reasoning: For patients under 55 years with **Stage 2 hypertension** (BP ≥ 160/100 mmHg), the first-line treatment is typically an ACE inhibitor unless there are contraindications. His age and absence of other risk factors point toward this approach.

*Note: If the patient were **≥55 years** or **Afro-Caribbean of any age**, the first-line treatment would be a **calcium channel blocker** (CCB).*

Scenario 5:

Patient: An 82-year-old woman presents with a clinic blood pressure of 145/90 mmHg. She has mild frailty but no history of cardiovascular disease, diabetes, or renal impairment.

Answer: **Individualized treatment** based on overall health, considering her frailty.

Reasoning: For patients over 80 years old, treatment decisions should be personalized. In this case, the patient's frailty means that the risks and benefits of medication need careful consideration, and lifestyle modifications might be a reasonable alternative if her health allows.

Scenario 6:

Patient: A 58-year-old man presents with a clinic blood pressure of 160/100 mmHg. He has no significant medical history and is of Afro-Caribbean descent.

Answer: Start with a **calcium channel blocker** (e.g., **Amlodipine**, **Diltiazem**).

Reasoning: For patients **≥55 years** or **Afro-Caribbean of any age**, the first-line treatment for Stage 2 hypertension is a calcium channel blocker. This is because CCBs have been shown to be more effective in these populations compared to ACE inhibitors or ARBs.

Scenario 7:

Patient: A 52-year-old woman started on **Ramipril** (ACE inhibitor) for her **Stage 2 hypertension**. After 3 months, her blood pressure remained elevated, so her doctor added **Amlodipine** (calcium channel blocker). However, she developed significant ankle swelling after starting the CCB.

Answer: Stop the **calcium channel blocker** due to the side effects and replace it with a **thiazide-like diuretic** (e.g., **Indapamide**, **Chlorthalidone**).

Reasoning: **Ankle swelling** (peripheral edema) is a common side effect of calcium channel blockers. In this case, the CCB should be discontinued and replaced with a thiazide-like diuretic.

The four most common side effects of calcium channel blockers are:

1. **Peripheral edema** (ankle swelling).
2. **Gingival hyperplasia** (gum overgrowth).

3. **Headache**.
4. **Flushing**.

Key 175

Management of Hyperkalemia in Hypertensive Patients on ACE Inhibitors

Overview:

A 58-year-old man with a history of well-controlled hypertension on **Ramipril** (an ACE inhibitor) presents for a routine follow-up. Recent blood tests show a potassium level of **6.0 mmol/L** (normal: 3.5-5.0 mmol/L), indicating moderate **hyperkalemia**. The patient is currently asymptomatic, with normal renal function and no significant past medical history.

Next Step in Management:

The most appropriate next step is to **arrange an ECG** to check for any electrocardiographic changes that may indicate life-threatening arrhythmias due to hyperkalemia.

Explanation:

Even though the potassium level of **6.0 mmol/L** represents moderate hyperkalemia, the patient is asymptomatic. An ECG is necessary to assess for changes such as:

- **Peaked T waves.**
- **Widened QRS complex.**
- **Prolonged PR interval.**

These changes can indicate a risk for life-threatening arrhythmias.

If ECG abnormalities are present, urgent treatment with **calcium gluconate** may be required to stabilize the cardiac membrane.

Post-ECG Management:

1. If ECG is Normal (No Significant Changes):

- **Stop or reduce Ramipril**: ACE inhibitors like **Ramipril** can increase potassium levels by reducing aldosterone activity, which promotes potassium excretion. Discontinuing or reducing Ramipril can help prevent further hyperkalemia.

- **Do not switch to an ARB**: ARBs (e.g., **Losartan**, **Candesartan**) also increase potassium levels by a similar mechanism and should be avoided in patients with hyperkalemia.
- **Switch to an alternative antihypertensive**:
  - **Calcium channel blockers (CCBs)** (e.g., **Amlodipine**) or
  - **Thiazide-like diuretics** (e.g., **Indapamide**) can be considered to control hypertension while promoting potassium excretion.
- **Monitor potassium levels**: Recheck potassium levels after discontinuing or changing medications to ensure they normalize.

2. If ECG Shows Abnormalities (e.g., Peaked T Waves, Widened QRS, Prolonged PR Interval):

- **Administer intravenous calcium gluconate**: This stabilizes the cardiac membranes and reduces the risk of arrhythmias.
- **Give treatments to lower potassium levels**:
  - **Insulin and dextrose**: To shift potassium into the cells.
  - **Nebulized salbutamol**: Another method to shift potassium intracellularly.
  - **Sodium bicarbonate**: If acidosis is present, it helps reduce serum potassium levels.

- **Stop Ramipril**: Discontinue ACE inhibitors to prevent further increases in potassium levels.
- **Do not switch to an ARB**: ARBs should also be avoided as they pose a similar risk of hyperkalemia.
- **Close monitoring**: Regular monitoring of **ECG** and **potassium levels** is crucial to track improvement and prevent complications.

Summary:

First, arrange an ECG, then in both scenarios (normal or abnormal ECG):

- **Discontinue ACE inhibitors** like Ramipril.
- **Avoid ARBs**, as they also contribute to hyperkalemia.

  (So, both ACE inhibitors and ARBs can cause hyperkalemia).

- **Switch to safer alternatives** such as **calcium channel blockers** or **thiazide-like diuretics**.
- Continue monitoring **renal function** and **potassium levels** to ensure effective management of hyperkalemia and maintain blood pressure control.
